THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—38TH YEAR. SYDNEY, SATURDAY, OCTOBER 20, 1951. No. 16.

Table of Contents.

CONGENITAL PULMONARY VALVULAR STENOSIS.

By R. B. BLACKET, A. JEAN PALMER[1] and E. J. HALLIDAY,
From the Hallstrom Institute of Cardiology, Royal Prince Alfred Hospital, Sydney.

CONGENITAL STENOSIS of the pulmonary valve without overriding of the aorta or ventricular septal defect was recognized by Peacock (1859). It occurred 23 times in the 272 hearts studied by Keith (1909), and 25 times in Abbott's series of 1000 malformed hearts (1936). Of her subjects, 16 had a patent *foramen ovale*. Currens, Kinney and White (1945) observed three cases during life confirmed at autopsy and found reports of eight more in the pathological records of three Boston hospitals. Of the 11 subjects, five had a patent *foramen ovale*. They remarked that according to Abbott's findings, at least two patients in seven with pulmonary stenosis will not have a ventricular septal defect, a fact not adequately recognized even among those particularly interested in congenital heart disease. In 1949 Allanby and Campbell reported clinical and post-mortem findings in nine cases of pulmonary stenosis with intact ventricular septum, and they pointed out that although this malformation is much less common than the tetralogy of Fallot, it occurs frequently enough to make its recognition important.

One must stress at the outset the importance of the *foramen ovale* or other defect of the interauricular septum in the clinical picture, a fact recently emphasized by Selzer and others (1949). Three clinical syndromes have been defined. In the first a large interauricular communication dates from infancy, and the clinical picture with cyanosis was described by Fallot (1888) as a trilogy to be distinguished from his better known tetralogy, which it closely resembles. In the second syndrome the *foramen ovale* closes normally in infancy and cyanosis is absent until the late stage of the disease, when it is peripheral in origin. The third group is intermediate between the other two. At rest the *foramen ovale* is functionally closed, no blood flows through it and there is no cyanosis: but sooner or later in the course of the disease cyanosis appears first on exercise and later at rest as a result of "blowing" of the membrane covering the *foramen ovale*.

Many examples of the third group are indistinguishable clinically or at catheterization from the second group, and it has become the custom to refer to acyanotic patients with a normal arterial oxygen saturation at rest as having uncomplicated or pure pulmonary stenosis. If patency of the *foramen ovale* is considered a complication, as we feel it should be, then these clinical terms must always be used with the reservation that there is at present no way of defining the anatomical state of the *foramen ovale* or its covering membrane; one can merely state that at the time of study no blood was passing through it.

In recent years the diagnosis of pulmonary valvular stenosis has been facilitated by the technique of cardiac catheterization. Three cases discovered in this way were reported by Pollack, Taylor, Odel and Burchell (1948), and since then Greene *et alii* (1949) and Dow *et alii* (1950) have published details of 13 more cases together with exhaustive reviews of previous work.

The present paper is derived from the study of 11 patients, and it is our belief that the diagnosis, confirmed in these cases by cardiac catheterization, can be made with reasonable certainty on clinical grounds alone. Nine of the patients were acyanotic, and therefore belong to the second and third groups defined above. The remaining two were examples of the third group, with only slight inconstant cyanosis. The patients were selected on the following physiological grounds: (1) the presence of a right ventricular systolic pressure above 40 millimetres of mercury (normal maximum, 30) and at least 15 milli-

[1] The early part of this work was carried out under the Marion Clare Reddall Research Scholarship, Department of Medicine, University of Sydney.

metres of mercury greater than the systolic pressure in the pulmonary artery; (ii) no significant difference in the oxygen content of blood from the superior *vena cava*, the right auricle, the right ventricle and the pulmonary artery; (iii) normal arterial oxygen saturation; the one exception to this will be discussed later.

In two of the earlier cases we were unable to advance the catheter beyond the outflow tract of the right ventricle; the remainder of the findings, both clinical and functional, were in conformity, so these two patients have been included, although with some reservation. We have deliberately omitted four patients who had a difference of systolic pressure between the pulmonary artery and right ventricle of less than 15 millimetres of mercury; in two of these cases the right ventricular pressure was normal and in two only doubtfully elevated. Whether, in view of the pulmonary artery dilatation which existed in all four, one calls them cases of idiopathic dilatation or mild pulmonary stenosis, the clinical findings differ in some respects from the cases to be described here, and the malady is so mild that no treatment is necessary. For brevity only three case histories are given in their entirety; they illustrate the common features of the disease.

Reports of Cases.

CASE I.—A male patient, aged thirteen years, had been born at term. He was blue for a few hours after birth. He had an obscure illness at the age of fourteen months which left him weak and slightly blue at times. He began school at the age of five years and was unable to keep up at games because of fatigue. At seven years he had scarlet fever and the parents were told that he had a heart murmur. He is still unable to compete in strenuous games because of fatigue and dyspnœa. There are no other relevant data.

Physical examination showed the patient to be a boy of normal size without clubbing of the digits or cyanosis. Normal systolic pulsation was present in the root of the neck, and there was no chest deformity. The apex beat was in the fifth left intercostal space three and a half inches from the mid-line below the nipple; it was slightly more forceful than usual. There was a moderate right ventricular thrust between the apex beat and the sternum, and a faint systolic thrill was present to the left of the sternum, maximal in the second left intercostal space and just palpable in the neck. A systolic murmur was audible all over the heart, but maximal in the second left intercostal space (grade II to III), high pitched, moderately harsh and audible into the neck. There was a third heart sound at the apex and a split first sound down the left border of the sternum. Investigation of the pulmonary second sound showed grade I splitting, and the sound was of normal intensity. The blood pressure was 110 millimetres of mercury, systolic, and 65 millimetres, diastolic. The chest, abdomen and urine were normal. The red blood cells numbered 4,690,000 per cubic millimetre and the hæmoglobin value was 13·3 grammes *per centum*; the white blood cells numbered 4600 per cubic millimetre.

Fluoroscopic examination was carried out. In the postero-anterior view the heart was of normal size and activity. The lung fields and hilar vessels were normal and the pulmonary artery was enlarged ("+" to "++") and pulsated a little more freely than usual. On examination with a barium bolus the aorta was left-sided and the left auricle was not enlarged. In the second oblique view the cardiac silhouette was within normal limits.

The electrocardiogram showed sinus rhythm, normal *P*, *QRS* and *T* waves, and a mean axis deviation of +87°. The heart was in the vertical position. The precordial leads were normal. The angiocardiogram was taken in the postero-anterior and left oblique positions. The right auricle and ventricle were of normal size and the interventricular septum bulged normally towards the right ventricle. The first and second films (Figure XI) showed a jet effect at the pulmonary valve; the pulmonary artery was slightly dilated. Some dye still remained in the right ventricle five seconds after the injection, when the left side of the heart was well visualized. The findings at catheterization are summarized in Table I.

CASE II.—The patient was a male, aged two and a half years. The mother's pregnancy had been normal, and the baby was delivered by forceps. He was normal at birth and never blue. At the age of ten days he began to vomit, and at four weeks he underwent a Ramstedt's operation for pyloric stenosis; a heart murmur was noted and the heart was enlarged. Since the operation he had been perfectly well and had gained weight normally. He had a normal amount of energy and did not become more breathless than other children of his age. There had been no attacks of cyanosis. He had had a convulsive seizure six weeks earlier, attributed by his doctor to gastro-enteritis. He had one brother, aged five years, alive and well, and the mother was well. The father had had acute nephritis and rheumatic fever.

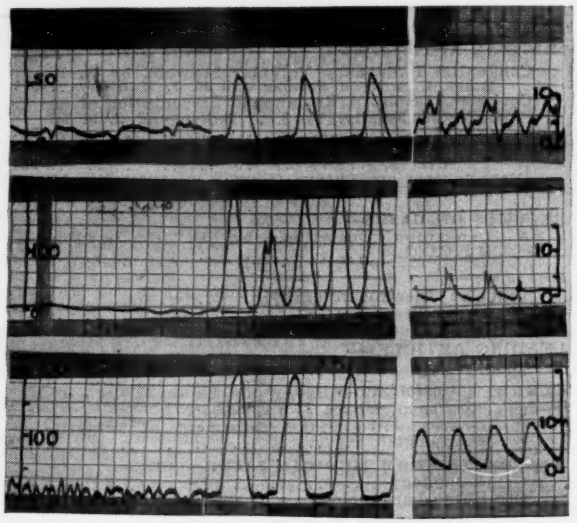

FIGURE I.

Partly retouched. Pressure pulse tracings in Cases I, II and III. In each tracing the curves were obtained from the pulmonary artery on the left, the right ventricle and the right auricle. The upper tracing shows the characteristic loss of the normal pulse contour in the pulmonary artery with an elevated systolic pressure in the right ventricle and a normal auricular tracing. In the second and third cases with more severe stenosis, the pulmonary artery tracing is even more damped by the stenosis than in the first, and the right ventricular systolic pressure is greatly elevated and is above the systemic levels. The auricular curves are abnormal owing to increased auricular contractions.

On examination the patient was a healthy looking child, normally developed, without cyanosis or clubbing of the fingers. His pulse rate was 110 per minute and the pulse was regular. The brachial blood pressure was 120 millimetres of mercury, systolic, and 80 millimetres, diastolic. The femoral pulses were normal, and so were the neck pulses. His chest was not deformed. The apex beat was in the fifth left intercostal space in the anterior axillary line, and was tapping in character. A faint systolic thrill was palpable in the second left intercostal space, and a systolic impulse was palpable over the rest of the precordium. The first sound in the pulmonary area was normal, but overshadowed by a loud, harsh, systolic murmur, maximal in the first and second left intercostal spaces, but heard all over the precordium, up to the left clavicle and at both lung bases posteriorly, in the left better than in the right. The second pulmonary sound was diminished and pure. There was a high-pitched, squeaky murmur (grade II), apparently exocardial, best heard in the third and fourth left intercostal spaces out towards the cardiac apex. No diastolic murmur was detected. A third heart sound was heard at the apex with the child supine. The lungs were normal. On the abdomen there was a Ramstedt's scar. The liver and spleen were not palpable, and the urine was normal. A blood count gave the following information: the red blood cells numbered 5,240,000 per cubic millimetre, the hæmoglobin value was 14·0 grammes *per centum*, the colour index was 0·9, and the white cells numbered 8000 per cubic millimetre and were of normal distribution.

On fluoroscopic examination the heart was greatly enlarged to the right and left and very quiet. The aorta was left-sided and about normal in size. The pulmonary artery was greatly enlarged and quiet. The pulmonary arteries on both sides were well marked but did not pulsate.

ILLUSTRATIONS TO THE ARTICLE BY R. B. BLACKET, A. JEAN PALMER AND E. J. HALLIDAY.

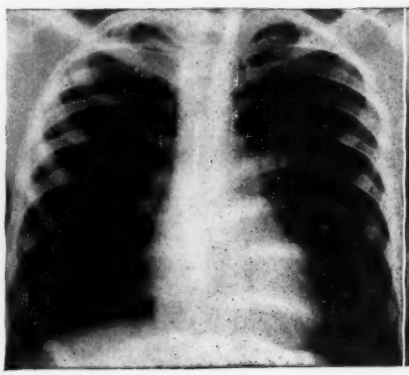

FIGURE II.

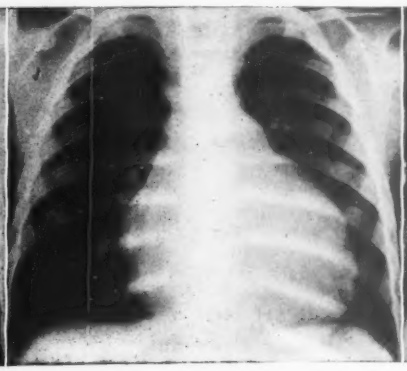

FIGURE IIIA.

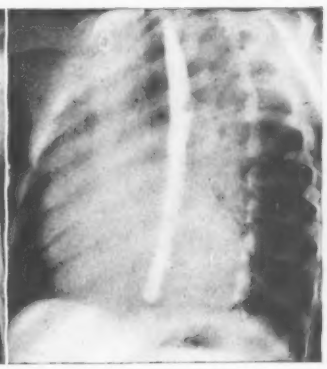

FIGURE IIIB.

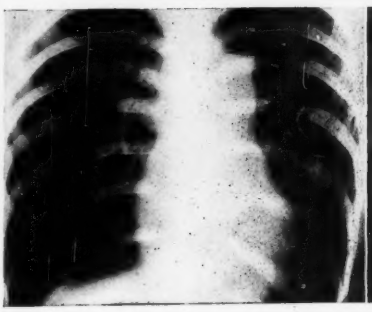

FIGURE IV.

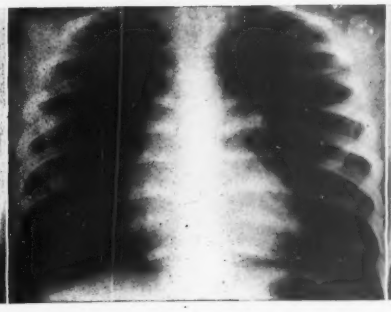

FIGURE V.

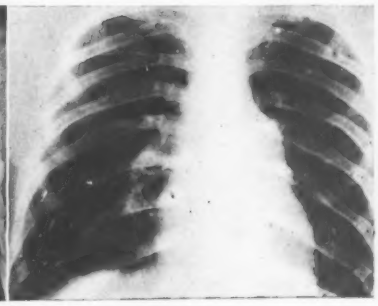

FIGURE X.

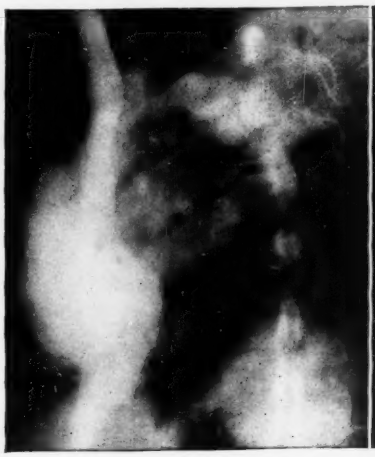

FIGURE XI.

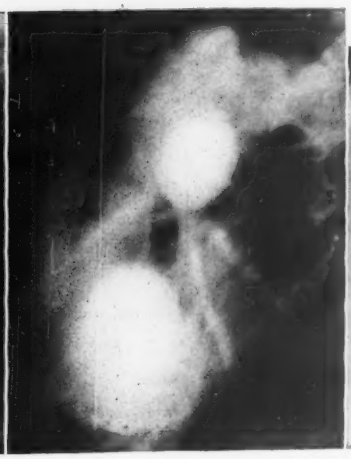

FIGURE XII.

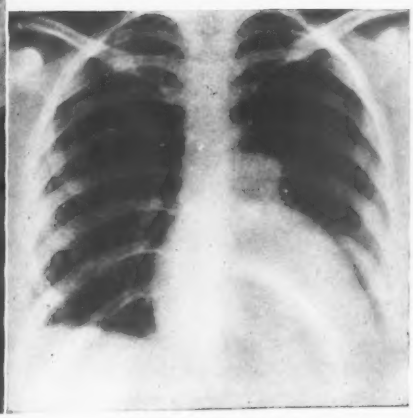

FIGURE XIII.

ILLUSTRATIONS TO THE ARTICLE BY S. J. BAKER AND J. J. M. O'NEILL

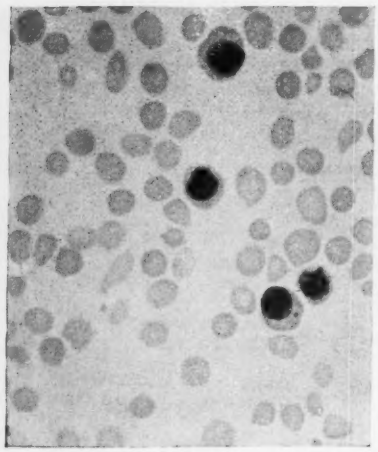

FIGURE II.

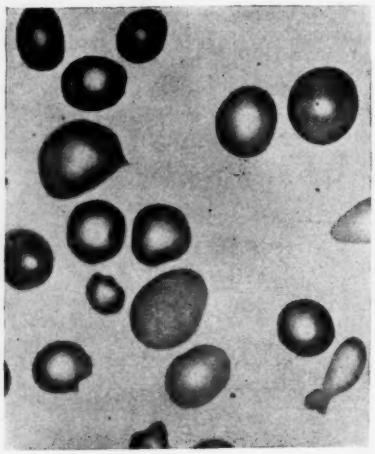

FIGURE III.

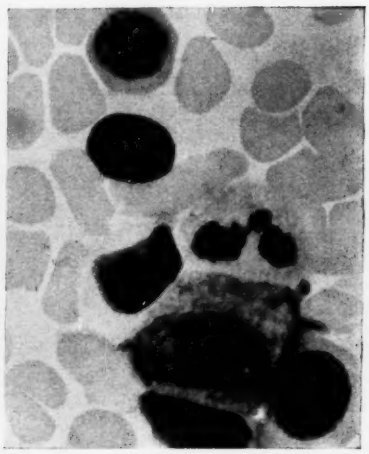

FIGURE IV.

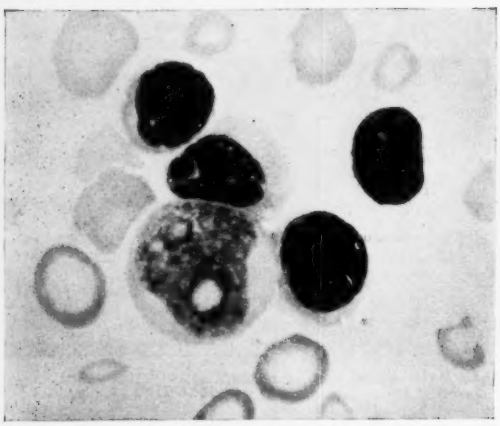

FIGURE V.

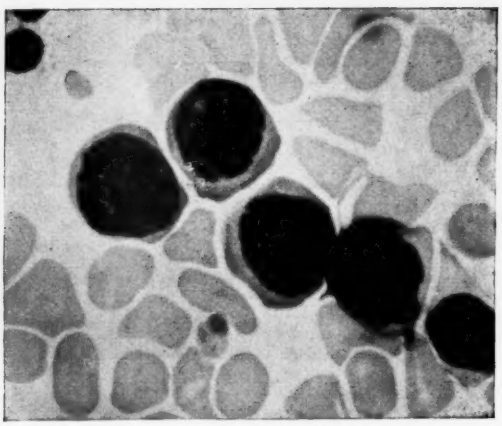

FIGURE VI.

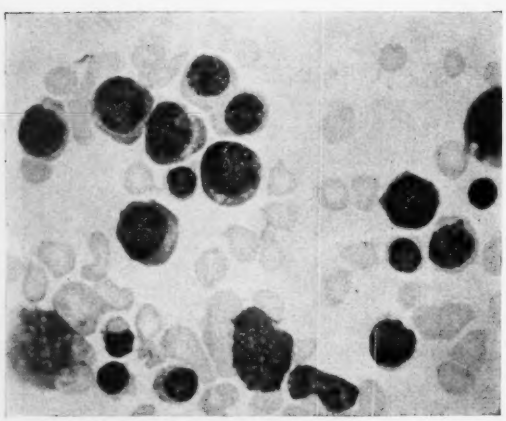

FIGURE VII.

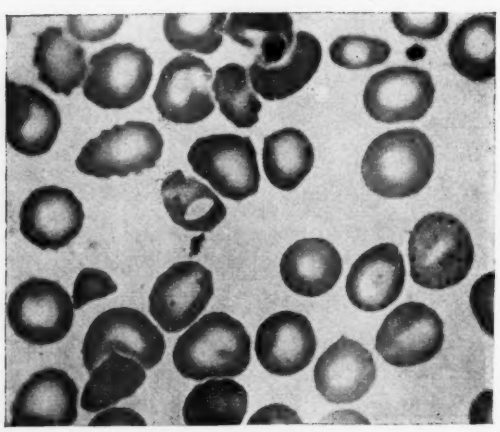

FIGURE VIII.

The lung fields were normal. The heart was only slightly enlarged to the right, but was greatly enlarged to the left, and the left border was convex. In the right oblique view the retrocardiac space was clear and the left auricle was not enlarged. In the left oblique view the right ventricle was much enlarged, and the posterior border of the heart was far from clearing the vertebral column in the 45° position.

The electrocardiogram showed right axis deviation with an *S* wave in lead I, and *Q* and inverted *T* waves with

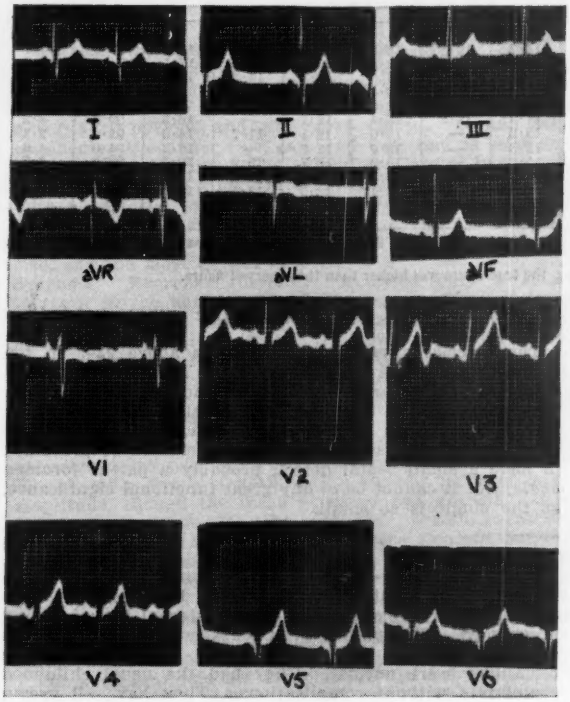

FIGURE VI.
Normal electrocardiogram (Case I).

slight depression of the *S-T* segment in lead III. The *P* waves in lead II were spiked and measured 3·5 millimetres in height. The heart was in the vertical position. There was deep inversion of *T* waves and depression of the *S-T* segments in leads V_1 and V_2. The transitional zone was about lead V_3. Pronounced right ventricular hypertrophy with clockwise rotation was present.

The catheterization findings appear in Table I.

CASE III.—A male subject, aged twenty-seven years, had always been well and had led a normal, fairly strenuous life as a mechanic. A few months earlier he had noticed a throbbing feeling in the epigastrium and throat, coming on after exertion or excitement and passing off quickly with rest. He had no pain, and save for slight shortness of breath on exertion, which had appeared only lately, he felt well. His past and family history was normal.

On examination, the patient was seen to be a somewhat underweight, thin but muscular man of good colour, without cyanosis or clubbing of the digits. The pulses were normal; the brachial blood pressure was 125 millimetres of mercury, systolic, and 75 millimetres, diastolic. There was pronounced venous pulsation at the root of the neck occurring immediately before the first heart sound. More laterally a small diastolic venous wave was seen. The venous pressure was normal even after exercise. The liver was of normal size and did not pulsate. There was some prominence of the left side of the chest. The apex beat was barely palpable with the patient in the supine position, but was more easily palpable with the patient leaning forward, in the fifth left intercostal space four inches from the mid-line. It was tapping in character. Between the apex beat and the sternum there was a definite ventricular heave. A very faint thrill was present in the first, second and third left intercostal spaces, with dulness to percussion in the second left intercostal space up to one inch from the sternum. No dulness was present to the right of the sternum. A loud systolic murmur occupying the whole of systole was best heard in the second and first left intercostal spaces at the sternum, as well as all over the heart and in the back. In the third and fourth intercostal spaces the first sound was particularly loud. Just inside the mitral area was a third heart sound, louder after exercise. On one occasion a diastolic murmur was heard by another observer at the lower end of the sternum. The remainder of the examination gave normal findings. The hæmoglobin value was 14·4 grammes *per centum*; no cell count was made.

At fluoroscopic examination, in the postero-anterior view the transverse diameter was seen to be slightly increased. The right border was convex. The left border was very active and more convex than usual, and the apex was lifted. The pulmonary artery was greatly enlarged and quiet. The hilar vessels were normal in size and did not pulsate. The peripheral vessels were diminished. The left auricle was not enlarged in the right oblique view. In the left oblique view the right ventricle was enlarged and the left ventricle did not clear the vertebral column.

The electrocardiogram showed sinus rhythm at a rate of 70 per minute. There was pronounced right axis deviation in the standard leads, and the *P* waves measured 3·5 millimetres and were spiked in lead II. The heart was vertical and was rotated clockwise. The precordial leads showed extreme right ventricular hypertrophy with a strain pattern in leads V_1 to V_6.

The angiocardiogram was taken in the left oblique view. It showed dilatation of the right auricle and ventricle and bulging of the interventricular septum to the left. The pulmonary artery was greatly dilated. The right ventricle was still opacified after six seconds, as was the pulmonary artery. The left side of the heart had filled more slowly than usual. The five-second film (Figure XII) showed a normally formed outflow tract of the same diameter as the proximal part of the pulmonary artery, from which it was separated by a transverse partition with a hole in the centre, taken to be the pulmonary valve. The pulmonary artery began abruptly at the valvular partition previously noticed, and the outflow tract was then practically clear of dye. These appearances were taken to indicate pulmonary valvular stenosis. The catheterization findings are shown in Table I.

Physiological Findings.

The methods used in catheterization differed in no significant detail from those of Cournand, Baldwin and Himmelstein (1949). In the earlier cases only mean pressures were measured by the use of a saline manometer. In the remainder strain gauges (Lambert and Jones, 1948) or an electromanometer were used (Rappoport and Sarnoff, 1949). Zero point for pressures was five centimetres behind the sternal angle in adults and a smaller proportional amount for children. The oxygen consumption was measured in only half the cases, and the calculated cardiac outputs will be referred to in the text. Cases I, II, V, VII, VIII and IX were studied with the patients under rectally induced "Avertin" anæsthesia. The results for the 11 cases are summarized in Table I.

The ventricular pressures in millimetres of mercury ranged from 60, systolic, and seven, diastolic, in Case V, to 171, systolic, and five, diastolic, in Case III (Figure I). The right auricular mean pressures and right ventricular end diastolic pressures were within normal limits in all cases. The mean pulmonary arterial pressure was measured in nine cases and was normal in seven; in Case IV it was seven and in Case X four millimetres of mercury, both low figures. In most of the cases the pulmonary arterial pressure curve was so damped by the stenosis that only mean figures could be read (Figure IB). The pressure taken in the outflow tract of the right ventricle immediately after withdrawal from the pulmonary artery was sometimes considerably higher than that found a few minutes later in the same region. This was ascribed to blocking of the stenosed orifice by the catheter. In Case II it fell from 164 millimetres of mercury, systolic, and three millimetres, diastolic, to 119 millimetres of mercury, systolic, and two millimetres, diastolic, and in Case III from 190 millimetres of mercury, systolic, and

TABLE I.
Catheterization Findings.

Case Number.	Age. (Yrs.)	Sex.	Pressures. (Millimetres of Mercury.)					Oxygen Content. (Volumes per 100 Millilitres of Blood).								
			R.A.[1] Mean.	R.V.[1]		P.A.[1]		B.A.[1]	S.V.C.[1]	I.V.C.[1]	R.A.[1]	R.V.[1]	P.A.[1]	Arterial.	Arterial. (Percentage Saturation.)	A.-V.[1] Oxygen Difference. (Volumes *per Centum*.)
				Systolic/Diastolic.	Mean.	Systolic/Diastolic.	Mean.									
I	13	M.	4	60/7	14	18/10	12	110/65	14·2	13·8	13·7	13·2	14·2	18·9	97·0	4·7
II	2½	M.	2	119/2	41	—	8	120/80	13·1	14·2	12·2	12·3	12·2	17·1	94·6	4·9
III	27	M.	5	171/5	60	20/5	13	130/80	13·1	13·9	13·6	13·7	14·3	18·9	98·6	4·6
IV	20	F.	1	102/3	31	—	7	94/52	10·9	—	10·4	10·4	10·9	18·1	96·2	7·4
V	13	F.	—	—	35	—	10	130/80	12·1	14·8	12·0	12·5	12·2	16·7	95·2	4·5
VI	7	M.	4	—	35	—	—	120/70	11·3	13·2	12·4	12·1	—	16·3	99·3	4·2
VII	7	M.	5	73/6	36	—	19	80/55	13·2	—	12·0	12·1	11·7	14·0	93·4	2·3
VIII	8	M.	4	—	39	—	—	105/60	13·8	—	13·7	13·7	—	18·3	94·0	4·6
IX	20	M.	7	70/8	25	11/6	9	118/68	15·3	—	15·9	15·7	15·9	20·6	98·0	4·7
X[2]	18	F.	2	>100/2	>40	6/2	4	108/64	12·7	14·9	12·0	11·9	11·4	18·1	91·2	6·7
XI	23	F.	3	55/65	13	—	12	115/68	12·9	—	13·1	13·5	13·5	17·6	97·2	4·1
				3												

[1] R.A., right auricle; R.V., right ventricle; P.A., pulmonary artery; B.A., brachial artery; S.V.C., superior *vena cava*; I.V.C., inferior *vena cava*; A.-V., arterio-venous.

[2] In Case X good pressure tracings were not obtained from the right ventricle; the true figure was higher than the observed figure.

five millimetres, diastolic, to 171 millimetres of mercury, systolic, and five millimetres, diastolic. In the last-mentioned case leg raising for two minutes raised the right ventricular pressure to 220 millimetres of mercury, systolic, and three millimetres, diastolic, with a return to normal in six minutes. We have not carried out this exercise test in all cases, though it could perhaps prove useful in demonstrating mild stenosis when the pressure differences are equivocal; the risk of ventricular arrhythmia, though not great, renders the procedure somewhat suspect.

The mean right auricular pressure was normal in all the cases. In four of them the form of the auricular pressure tracing was abnormal. Although not shown graphically, it was apparent by auscultation when the catheter was connected to the electromanometer with a direct writing recorder that the main deflection preceded the first heart sound and was due almost certainly to auricular contraction.

The oxygen content of the mixed venous blood did not vary significantly from the great veins to the pulmonary artery in any of the cases. The arterio-venous oxygen difference was low in Case VII and this was attributed to the lightness of the anæsthesia. In eight of the cases the difference ranged from 4·1 to 4·9 volumes *per centum* and was therefore normal. In Cases IV and X it was 7·4 and 6·7 volumes *per centum* respectively and was therefore raised. These two patients were both handicapped and had low cardiac outputs. The respective cardiac indices were 2·27 and 2·17 litres per square metre of body surface per minute, which are both below the normal of 2·70 to 3·50 litres per minute (Harvey *et alii*, 1949). In Cases I, III, IV, IX and XI the cardiac outputs and indices were normal.

The arterial oxygen saturation was above 93% in ten of the 11 cases, and for the method we used this is normal. In Cases III, IV and IX there was no fall in saturation with exercise. In Case X the arterial oxygen saturation was 91·2%, which fell to 87·5% when the patient was exercised to exhaustion. She must therefore be presumed to have a small septal defect, probably a patent *foramen ovale*; but it cannot be of any great functional significance, as the shunt is so small.

Clinical Findings.

The relevant cardiac findings are summarized in Table II. All the patients came of healthy families, and there was no history of rubella or other maternal illness during pregnancy. The patient in Case I was cyanosed for a short time after birth, but so far as is known the remainder were normal. They had the usual childhood complaints without complications. They have all grown normally, even the patient in Case X, who was so handicapped as to be unable to attend school. Three of the patients had associated malformations. In Case IV there was bilateral *talipes varus* of the feet. The seventh patient, as well as being obese, had extreme myopia, rather small low-set ears, an undescended testicle and partial syndactyly of the second and third toes of both feet. The patient in Case X had thoracic scoliosis. The murmur was found very early in life in several instances. If looked for, it is probably present from birth.

The degree of disability varied greatly, and it is at once apparent that pulmonary stenosis may be a very mild or

TABLE II.
Summary of the Most Valuable Cardiac Signs.

Case Number.	Character of Apex Beat.	Precordial Thrust.	Site of Thrill.	Site of Murmur.	First Sound Left Sternal Border.	Pulmonary Second Sound.
I	Tapping.	+	Second L.I.C.S.[1]	Second L.I.C.S. up to neck.	Normal.	Normally split.
II	Tapping.	++	Second L.I.C.S.	Second L.I.C.S. to clavicle and back.	Normal.	Diminished.
III	Faint tapping.	+++	Second L.I.C.S. Very faint.	Second L.I.C.S. all over precordium.	Accentuated.	Diminished.
IV	Tapping.	++	First to third L.I.C.S.	Second L.I.C.S.	Not stated.	Faint.
V	Tapping.	++	First and second L.I.C.S. almost equally.	First and second L.I.C.S.	Accentuated.	Diminished.
VI	Normal.	+	Second L.I.C.S.	Second L.I.C.S.	Not stated.	Normal.
VII	Normal.	+	First and second L.I.C.S.	Second L.I.C.S. up to clavicle and back.	Accentuated.	Normal.
VIII	Normal.	Weak.	Third L.I.C.S.	Second L.I.C.S. up to clavicle.	Normal.	Diminished.
IX	Not stated.	+	Second and third L.I.C.S.	Second L.I.C.S.	Not stated.	Split.
X	Not palpable.	Absent.	Second L.I.C.S.	Second L.I.C.S. up to clavicle and back.	Accentuated.	Greatly diminished.
XI	Tapping.	+	Second L.I.C.S.	Second L.I.C.S.	Normal.	Diminished.

[1] L.I.C.S. = left intercostal space.

most severe handicap; in the mildest cases the subjects have no disability at all. Fatigue and dyspnœa on exertion have been frequent complaints, and these should direct attention to the circulation. Two patients had *angina pectoris*. Squatting is most unusual, but occurred in Case X. The disability was roughly proportional to the right ventricular pressure, but there are exceptions. A mean pressure of more than 40 millimetres of mercury is probably always associated with diminished exercise tolerance, but the pressure may be much higher without producing great incapacity (see Case III). By contrast, the patient in Case IV, with a mean pressure of 31 millimetres of mercury, was severely handicapped.

Cyanosis was seen in two patients (Cases VIII and X), and was stated to have occurred in Case I in infancy, but not since. In the two cases in which it was observed, the cyanosis varied considerably from day to day. It was never very obvious and was often absent. It has been confined to the finger tips, ears and toes, and is seen less frequently in the lips. The patient in Case X had a low cardiac output and a high arterio-venous oxygen difference, with an arterial oxygen saturation slightly below normal at rest with a further slight fall on exercise. Furthermore, the cyanosis showed little if any deepening on exercise, and the most noticeable features were the pallor and dyspnœa. Exercise, therefore, produced only a small increase in the shunt, and in this respect these patients differ greatly from those with the tetralogy of Fallot. The most cyanosed patient (Case VIII) was unfortunately not given an exercise test, and we were surprised to find that he had normal arterial oxygen saturation at rest. He had good exercise tolerance, and it is probable that the cyanosis was peripheral in origin.

Polycythæmia or unequivocal clubbing of the fingers was not seen in any patient. Three patients (Cases III, IV and X), all with severe stenosis, had a jugular pulse of large amplitude, though the mean pressure was normal. In a fourth case (Case II) the auricular pressure tracing showed the characteristic pulse of severe stenosis, but the abnormal wave was not thought to be visible in the neck. The main wave precedes the first heart sound, and we agree with Wood (1950) that it is a sign of severe disease. It is often accompanied by a pulsating liver; this was observed in all three of Engle and Taussig's cyanotic cases. All such cases are severe.

The apex beat was not conspicuous. In the majority of cases it was described as normal, and in the remainder it was a gentle ill-sustained tip. Of more value was the systolic thrust, which could usually be felt between the apex and sternum over the right ventricle. This was particularly obvious in Case III. This patient had the highest right ventricular pressure, and though it was not so pronounced in the others, it is a sign which we have come to value. It is not specific and is seen in most cases of congenital heart disease when the right ventricle is enlarged.

The systolic thrill has been found in every case, though it has not always been obvious with the patient in the supine position, or indeed present at every examination unless the patient is exercised. It was felt most frequently in the second left intercostal space near the sternum, but in two cases it was felt equally well in the first. It may be felt as far down as the fourth intercostal space, but we think this unusual. At times it is perceptible at the root of the neck. The systolic murmur is nearly always loudest and earliest in the second or even the first left intercostal space. We have found it loudest in the third intercostal space, but not as yet in the fourth. Moreover, no patient with a systolic murmur maximal in the third and fourth intercostal spaces has had radiological or cardiographic evidence of pulmonary valvular stenosis without a septal defect, and none has been shown at catheterization to have this lesion. The murmur lasts throughout systole, is loud and harsh especially in severe cases, and is produced at the valve. The propagation is characteristically upwards towards the left clavicle; because of its loudness the murmur may be heard well in the neck or better in the back, particularly on the left side. It may be followed by the short diastolic murmur of pulmonary insufficiency, but this did not occur in any of our cases.

The pulmonary second sound is diminished or even absent in severe cases, and in the less severe it is normal and to the ear and on the phonocardiogram it may be split.

Hitherto little attention has been paid to the first heart sound, though it has often been reported as accentuated or split in case histories. Save in the milder cases, it

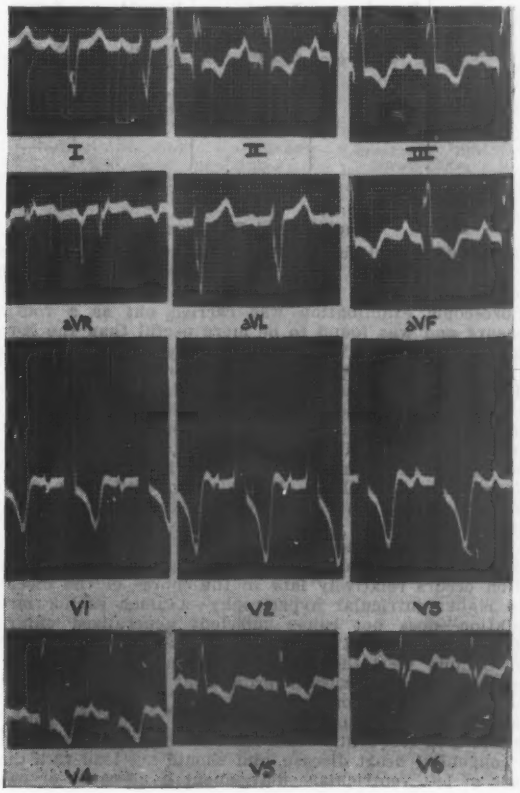

FIGURE VII.

Electrocardiogram in Case X. The *P-R* interval is 0·22 second. The *P* waves in lead II are abnormally tall, and the duration of the *QRS* complex is 0·15 second. The heart is vertical, and lead AVF resembles leads V_4 and V_5. The precordial leads show right bundle branch block due to extreme right ventricular hypertrophy, with characteristic deep inversion of the *T* waves in the leads derived chiefly from the right ventricular epicardium. A wide *S* wave appears in lead V_6, but no characteristic left ventricular epicardial tracing was obtained.

may be very loud, not in the pulmonary area, where it is obscured by the murmur, but in the third and fourth left intercostal spaces and out to the apex. It is not an illusion created by the diminution of the second sound, for it has been heard when the latter was normal. It is doubtless an expression of the abrupt and forceful contraction of the right ventricle, but it differs in quality from the accentuated first sound of mitral stenosis.

A third heart sound in early diastole was recorded at the apex in six cases. Three of these patients were children of an age at which the sound is frequently heard in normal hearts. Three were adults (Cases III, IV and X), all with severe disease, of whom one was a cardiac invalid and two, although active, had deteriorated recently. These findings support the view that this type of triple rhythm indicates actual or impending right heart failure (Evans, 1943).

TABLE III.

Radiological Features in 11 Cases of Pulmonary Stenosis.

Case Number.	Heart Size.[1]	Right Auricle.[1]	Right Ventricle.[1]	Left Auricle.[1]	Left Ventricle.[1]	Pulmonary Artery.[1]	Hilar Vessels.	Peripheral Lung Vascular Markings.
I	Normal.	Normal.	Normal.	Normal.	Normal.	Normal to +.	Normal.	Normal.
II	++	+	++	Normal.	++	++	Normal to small.	N. to d.[2]
III	+	+	+	Normal.	+	+++. Quiet.	Normal.	Diminished.
IV	++	+	++	Normal.	+	+	Small.	Diminished.
V	Normal.	Normal.	Normal to +.	Normal.	Normal.	++. Pulsates +.	Normal.	Normal.
VI	Normal.	Normal.	+	Normal.	Normal.	Normal to +.	Normal.	Normal.
VII	+	+	++	Normal.	Normal.	++	Normal.	N. to d.
VIII	Normal.	Normal.	Normal.	Normal.	Normal.	+. Pulsates +.	Left branch enlarged and pulsates.	Normal.
IX	Normal.	Normal.	+	Normal.	Normal.	++	Normal.	Normal.
X	+	+	+	Normal.	Normal.	+++	Small.	Small.
XI	Normal.	Normal.	Normal.	Normal.	Normal.	+	Left branch enlarged. Right normal.	Normal.

[1] "+", enlarged; "+ + +", grossly enlarged. [2] N. to d. = Normal to diminished.

Radiological Findings.

The radiological findings have recently been described by Healey, Dexter, Elkin and Sosman (1950). The findings in our own cases are summarized in Table III. Fluoroscopic examination was carried out as a routine measure and was found to be more useful than the X-ray films. Figure IV (Case III) shows the characteristic outline of the heart in the postero-anterior view.

Right ventricular enlargement was thought to be present in eight of the 11 cases. It was pronounced in Cases II and IV. Measurement of the cardio-thoracic ratio and comparison of the actual transverse diameter of the heart with that predicted from the patient's height and weight (Ungerleider and Clark, 1942) contributed little useful information, as the results were significantly abnormal only in those cases in which cardiac enlargement could be readily diagnosed by fluoroscopic examination. We suspect that in pulmonary stenosis right ventricular dilatation occurs relatively late in the course of the disease, and right ventricular hypertrophy—a much earlier manifestation—does not cause sufficient generalized enlargement to be reflected in the usual measurements. In the left oblique view the right ventricular enlargement was demonstrated by forward projection of the cardiac silhouette. In some of the cases there was also backward displacement of the posterior border of the cardiac shadow. This is seen frequently with right ventricular enlargement in congenital heart disease, and should not lead to a diagnosis of left ventricular enlargement if no cause or other signs can be found.

There was obvious right auricular dilatation in the severe cases, and on exploration with the catheter enlargement seemed to be present in some of the milder cases.

The infundibulum was thought to be slightly prominent in Cases III and X, and was recorded as normal in Cases I, V and XI. An example of extreme prominence is shown in Figure XIII.

The pulmonary artery was enlarged in nine of the 11 patients. The exceptions were children with mild to moderate stenosis only. Pulsation of the main pulmonary artery was commonly observed, though it was never impressive. In two patients (Cases VIII and XI) the dilatation and pulsation extended beyond the bifurcation to include the left main branch (Figure V). Unless the main branches of the pulmonary artery are dilated they do not pulsate.

The hilar vessels (save for dilatation of the left branch in two patients) and the vascularity of the peripheral lung fields were normal in six of the eleven cases. In two they were doubtfully diminished and in three definitely diminished. The left auricle was normal in size in all cases and the aorta was always left-sided; the left ventricle was assumed to be normal.

Angiocardiography.

Angiocardiography has been carried out in only four of the cases. All our patients have been exposed in the antero-posterior or left oblique position; so far we have not used the right oblique position. In pulmonary stenosis the findings have been as follows: (i) dilatation of the right auricle; (ii) delay in emptying of the right auricle and especially the right ventricle, which may be well seen even after six to eight seconds; (iii) dilatation of the right ventricle with bulging of the interventricular septum towards the left ventricle; (iv) in some cases demonstration of the constriction; (v) dilatation and delay in emptying of the main pulmonary artery with normal-sized branches; (vi) normal or diminished peripheral lung vasculature.

These findings confirm and extend those of Campbell and Hills (1950) and Engle and Taussig (1950).

In none of the patients in this series could we demonstrate a right-to-left shunt through the *foramen ovale*. In patients with pulmonary valvular stenosis and a large shunt it can usually be seen on the angiogram. Delay in emptying of the right ventricle without visualization of the aorta is good evidence of obstruction to flow from the right ventricle. Actual visualization of the constriction gives even better evidence. In Case I (Figure XI) the jet-like stream of the dye passing into the pulmonary artery could be seen, while in Case III close examination of the films showed the outline of the stenosed valve (Figure XII).

Electrocardiograms.

Electrocardiograms have been analysed according to the criteria of Wilson *et alii* (1944), which have been confirmed and extended by Myers, Klein and Stofer (1948). The most useful information is given in Table IV.

Standard Limb Leads.

Sinus rhythm was present in all cases, and the *P-R* interval in the more severe tended towards or was just above the upper limit of normal. The *P* waves were measured in lead II, and there was surprisingly good correlation between their height and the mean right ventricular pressure. Cases I, V, VI, VII, IX and XI were the mildest clinically, and at catheterization all these patients had *P* waves less than two millimetres high. In the remainder the condition was obviously more severe; the patients had higher right ventricular pressures and high peaked *P* waves varying from 2·4 to 3·5 millimetres in height. The conclusion is that while moderate stenosis and normal-sized *P* waves can coexist, abnormally high *P* waves are a sign of severe stenosis provided they are not due to the position of the heart. In the five cases in which such waves were present, there can be little doubt the right auricle was enlarged.

Right axis deviation in the standard leads occurred in all save Cases I and VI. In the former the axis deviation was just within normal limits, while in the latter a semi-horizontal heart with right ventricular hypertrophy resulted in a normal electrical axis. As this measurement depends so much on the position of the heart, it cannot

TABLE IV.
Electrocardiographic Findings.

Case Number.	Height of *P* Wave in Lead I (Millimetres).	Mean Axis Deviation.	Duration of *QRS*. (Seconds.)	R-S Ratio.		Intrinsicoid Deflection. (Seconds.)		Electrocardiographic Diagnosis.
				Lead V_1.	Lead V_6.	Lead V_1.	Lead V_6.	
I	1·2	+87°	0·07	$\frac{3·5}{8·0}=0·44$	$\frac{15·0}{2·5}=6·0$	0·026	0·047	Normal cardiogram.
II	3·3	+104°	0·11	$\frac{21·0}{0}=\infty$	$\frac{16·0}{14·0}=1·1$	0·066	0·024	Right ventricular hypertrophy.
III	3·5	+135°	0·11	$\frac{28·0}{0}=\infty$	$\frac{9·0}{11·5}=0·78$	0·070	0·028	Right ventricular hypertrophy.
IV	3·5	+144°	0·10	$\frac{39·5}{1·5}=26·3$	$\frac{0}{1·7}=0$	0·065	—	Right ventricular hypertrophy.
V	1·9	+124°	0·07	$\frac{9·3}{0}=\infty$	$\frac{2·2}{3·3}=3·09$	0·048	0·026	Incomplete right bundle branch block.
VI	1·5	+74°	0·06	$\frac{4·8}{1·9}=2·5$	$\frac{21·0}{5·5}=3·8$	0·038[1]	0·036	Incomplete right bundle branch block.
VII	1·5	+119°	0·09	$\frac{14·5}{2·5}=5·8$	$\frac{6·5}{3·4}=1·9$	0·063	0·038	Incomplete right bundle branch block.
VIII	2·4	+108°	0·08	$\frac{25·0}{0}=\infty$	$\frac{8·0}{5·5}=1·4$	0·052	0·028	Right ventricular hypertrophy.
IX	1·8	+120°	0·11	$\frac{23·5}{5·5}=4·3$	$\frac{12·0}{7·0}=1·7$	0·078	0·038	Incomplete right bundle branch block.
X	2·5	+139°	0·15	$\frac{31·0}{0}=\infty$	$\frac{6·0}{3·0}=2·0$	0·079	0·036	Right bundle branch block.
XI	1·6	+95°	0·06	$\frac{1·8}{0}=\infty$	$\frac{0·6}{0·2}=3$	Indeterminate.	0·024	Right ventricular hypertrophy.

[1] Intrinsicoid deflection measured in lead V_2R.

be expected to supply precise information about the right ventricle, nor has it done so in this series. In very mild stenosis the axis deviation is usually normal (Dow *et alii*, 1950).

The duration of the *QRS* complex in the standard leads was less than 0·09 second in five cases, 0·09 to 0·11 second in five cases, and 0·15 second in one case. Inversion of the *T* wave in leads II and III with or without *S-T* segment depression occurred in four severe cases. In another fairly severe case with cyanosis the *T* wave was rather low in lead II and inverted in lead III. In Cases I, V, VI, VII, IX and XI, which were less severe, the *ST* segments and *T* waves in the standard leads were normal. These patients also had normal *P* waves. Only three of them had a mean axis deviation of over 100; this confirms the findings of Myers, Klein and Stofer that the diagnosis of right ventricular hypertrophy cannot be made with certainty on the standard leads alone, although it may be suspected.

Unipolar Chest Leads.

The unipolar precordial leads have furnished the most direct and convincing evidence of right ventricular hypertrophy, and have the advantage over radiological examination in that they provide easily measurable variables. Furthermore, when the accepted criteria for right ventricular hypertrophy are satisfied, the method supplies highly reliable evidence for the diagnosis. On the other hand, ventricular hypertrophy may be present without the characteristic electrocardiographic signs. This we presume to be the case in our first patient whose cardiogram was normal, although lead V_2R was not obtained (Figure VI).

Of the remaining 10 patients, one had complete right bundle branch block, four incomplete right bundle branch block, and five right ventricular hypertrophy. Complete right bundle branch block was diagnosed on the basis of a *QRS* complex lasting 0·12 second or more, with tall notched *R* waves, delayed intrinsicoid deflection, absent *S* waves and inverted *T* waves in lead V_1, and a deep broad *S* wave in lead V_6 (Figure VII). Incomplete right bundle branch block was diagnosed in the presence of a *QRS* complex lasting less than 0·12 second (usually 0·09 to 0·11 second), with notching, double peaking or an *RSR* complex, small or absent *S* waves and delay in the intrinsicoid deflection in leads derived from the right ventricle, and a deep broad *S* wave in leads derived from the left ventricle (Figure VIII). The five patients with right ventricular hypertrophy in the electrocardiogram had tall *R* waves with a delay in the intrinsicoid deflection, absent *S* waves and inversion of the *T* waves in lead V_1, and a deep *S* wave in lead V_6. The duration of the *QRS* complex was less than 0·12 second (Figure IX). Although we have conformed to the accepted criteria for the diagnosis of right ventricular hypertrophy and partial or complete right bundle branch block, it is evident that we are dealing in all these cases with the same pathological process. Right bundle branch block has been reported only twice previously—by Blackford and Parker (1941) and by Durand and Metianu (1949). It was found in another of our patients, whose X-ray film is shown in Figure XIII. It has always occurred in severe cases, but not in the less severe, in which incomplete right bundle branch block or right ventricular hypertrophy is found.

It seemed likely at one stage that there would be a fairly precise correlation between the mean right ventricular pressure and the time of onset of the intrinsicoid deflection. Some relationship should exist, for right ventricular pressure must be a measure of the stenosis, while the intrinsicoid deflection is related to the muscle mass of the ventricle. However, two factors make its demonstration difficult: first the age of the patients, which cannot be controlled on account of the small number of patients, and secondly the fact that patients with pulmonary stenosis may show quite large variations in the right ventricular pressure on account of anxiety. In spite of these deficiencies measurement of the intrinsicoid deflection in lead V_1 does give a fairly good indication of the height of the right ventricular pressure and the severity of the disease.

In the four most severe cases (II, III, IV and X) the *S-T* segment abnormality seen in lead V_1 continued across the chest as far as leads V_5 to V_7 (Figures VII and IX). This pattern of extreme right ventricular enlargement is seldom seen in other forms of congenital heart disease and even less commonly in acquired disease. In the remaining cases with abnormal cardiograms the *T* wave inversion was seen only in lead VI, and the remaining precordial leads were normal save for the prominent *S* wave in leads V_5 and V_6.

Unipolar Limb Leads.

In three of the four severe cases lead AVR showed large inverted *P* waves and a *QR* complex. In the fourth (Case IV) this lead showed a tall *R* wave only. The *T* waves were inverted in two cases (Cases II and IV) and upright in two (Cases III and X). Lead AVL showed either upright or inverted *P* waves and an *RS* complex with an upright *T* wave. In Case IV lead AVL showed a *QR* complex with an upright *T* wave. The left leg lead resembled the right-sided chest leads. These findings were interpreted as pronounced clockwise rotation in vertical hearts with right ventricular hypertrophy.

In the less severe cases, similar though less pronounced patterns were found in leads AVR and AVL; lead AVF resembled the left ventricular epicardial lead.

In the mildest case there was a normal cardiogram and a vertical heart without clockwise rotation. Thus the heart was vertical in all cases and the degree of clockwise rotation depended largely on the severity of the stenosis.

In summary, in all cases save the very mild one the electrocardiograms were abnormal. Right ventricular hypertrophy or incomplete right bundle branch block was found with equal frequency, and the occurrence of one or the other did not seem to depend on the severity of the lesion. One case of complete right bundle branch block is added to the two already reported. This malformation, more than any other, regularly leads to a remarkable degree of right ventricular hypertrophy. This extreme hypertrophy is not confined to this malformation, for we have seen it electrocardiographically during life and confirmed at autopsy in patency of the *ductus arteriosus* with pulmonary hypertension, in auricular septal defect, in Fallot's tetralogy in an adult and in the Eisenmenger complex. It also occurs occasionally in mitral stenosis. Nevertheless, the finding of extreme right ventricular hypertrophy of the pattern illustrated should always suggest pulmonary stenosis.

Discussion.

Anatomy.

From our findings, pulmonary valvular stenosis without overriding of the aorta or ventricular septal defect appears to be not uncommon in congenital heart disease. Until recently the precise diagnosis of cardiac malformation has had to await autopsy. Current methods of diagnosis during life establish with reasonable certainty the presence of pulmonary valvular stenosis, but doubt remains as to their ability to demonstrate additional minor malformations of the heart. Patency of the *foramen ovale* may lead to a slight though significant degree of unsaturation of the arterial blood at rest and during exercise. It was found with certainty in only one patient, but must have been present, at least potentially, in some of the others. Recently Joly, Carlotti, Sicot and Piton have shown that in this malformation some patients with an arterial oxygen saturation at rest greater than 95%, and so normal, show a significant fall in saturation with exercise. We have not yet found such a case; but Burchell and Wood (1950) remark that "one cannot assume complete anatomical closure of the *foramen ovale* on the basis of normal arterial oxygen saturation". They do not give their evidence, but presumably they have been fortunate enough to pass the catheter through the *foramen ovale*. It can be assumed that an exercise test will detect patency of the *foramen ovale* in some cases; there is no proof that it will do so in all cases of pulmonary stenosis in which the foramen is patent.

A defect in the interventricular septum may also lead to difficulty. Two situations may exist. With mild pulmonary stenosis resulting in a moderate rise in right ventricular pressure the shunt will presumably be from left to right and will depend for its detection on the finding of a higher oxygen content of blood in the outflow tract and pulmonary artery than in the body of the right ventricle and the right auricle. We have found two such patients (not included in this series); in a third the clinical diagnosis was pulmonary stenosis. At catheterization the catheter passed through a ventricular septal defect into the aorta, but was not introduced into the pulmonary artery. The right ventricular pressure was moderately raised without evidence of a shunt in either direction. It is therefore possible that a small ventricular septal defect may be missed at catheterization. Presumably an exercise test may help by producing a reversal of the shunt and a fall in arterial saturation; we have not found such a case. With severe pulmonary stenosis and a ventricular septal defect the shunt is from right to left and the patient is cyanosed. The defect can be demonstrated either at catheterization or by angiocardiography, but cannot be distinguished on these grounds from Fallot's tetralogy with overriding of the aorta.

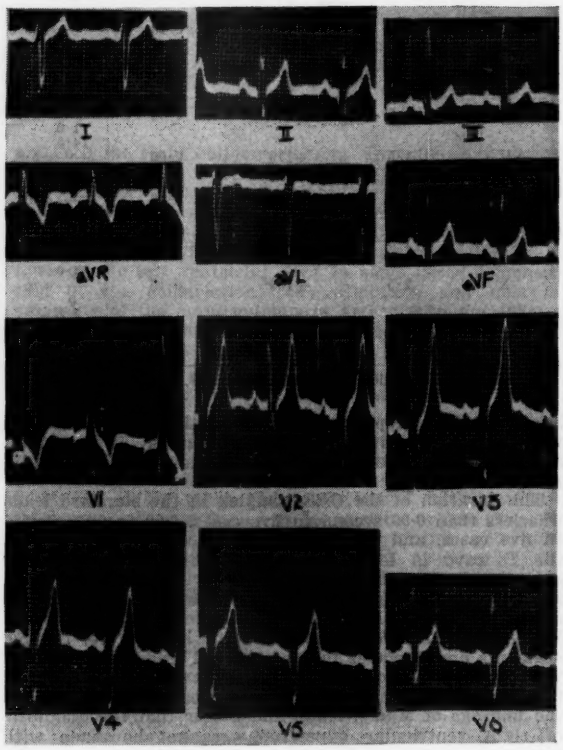

FIGURE VIII.
Electrocardiogram in Case IX, showing incomplete right bundle branch block.

In his series of 272 congenitally abnormal hearts Keith found stenosis of the pulmonary valve due to fusion of the semilunar cusps 23 times. Of these hearts, four of the five which he was able to examine completely had in addition an "arrested condition of the infundibulum". To demonstrate this during life by catheterization or by angiocardiograms taken at a rate of one or two a second can be extremely difficult and at times impossible. The pressure tracing taken as the catheter is withdrawn from the pulmonary artery to the right ventricle in Fallot's tetralogy is sometimes indistinguishable from that seen in pure pulmonary valvular stenosis. The site at which the rise of pressure occurs cannot always be defined anatomically under the X-ray screen, nor can infallible conclusions be drawn from the movements of the catheter as it lies high in the outflow tract of the right ventricle. Pulmonary valvular stenosis occurs in typical Fallot's tetralogy with deformity of the infundibulum frequently

enough to make it probable that in some at least of the cases of "pure" pulmonary stenosis found during life some malformation of the infundibulum will be found. In two of our cases the catheter slipped quickly from the region of the pulmonary artery to the right auricle, and in Case X it was very difficult to keep it on the ventricular side of the tricuspid valve. We suspect, but have no proof, that both these patients have a deformity of the infundibulum. At present one can only affirm from past experience that the significant obstruction to the circulation in most cases of acyanotic pulmonary stenosis is at the valve, and that other complicating malformations are of no functional significance.

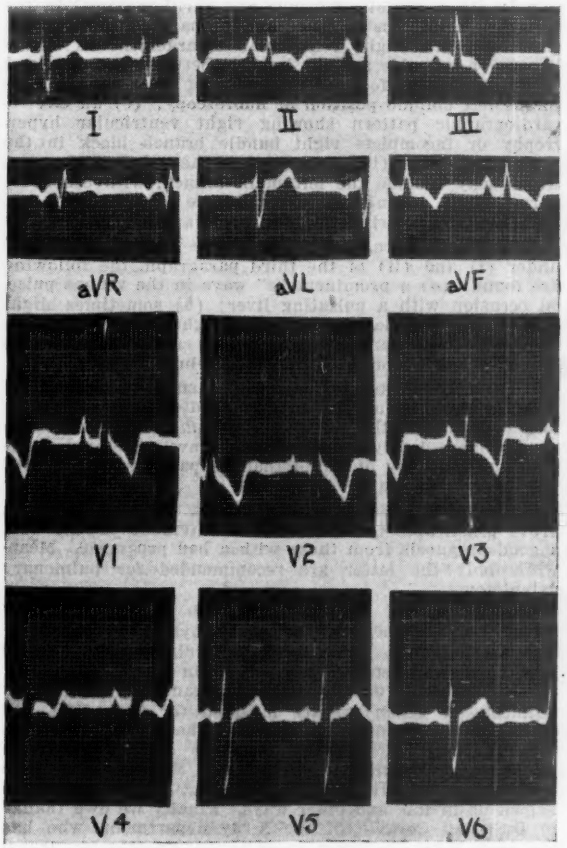

FIGURE IX.

Electrocardiogram in Case III. Similar to Figure VII, but showing right ventricular hypertrophy with a tall *R* wave and absent *S* wave in lead V_1 and a deep *S* wave in lead V_6.

Natural History.

The natural history of the disease has been considered in detail by Allanby and Campbell and by Engle and Taussig, and need only be summarized here. At birth the pulmonary valve leaflets are adherent at their edges, so as to form either a diaphragm with a hole a few millimetres in diameter in the centre or, less commonly, irregularly thickened and deformed leaflets with an irregular slit between them. The resistance to flow so created leads to hypertrophy of the right ventricle, which can then supply enough energy to the blood to overcome the resistance and maintain a normal perfusion pressure in the pulmonary circulation. The high rate of flow through the pulmonary valve produces the thrill and murmur, while turbulence beyond the valve usually leads to dilatation of the pulmonary artery. At rest compensation for the obstruction is complete and the majority of the infants grow normally to puberty and adult life. In some the obstruction is so great that death occurs in infancy or childhood, or if these are survived, growth is deficient. In childhood the majority can run about as well as, or almost as well as, healthy children. There is no reason to believe that the malformed valvular orifice ever becomes enlarged, unless bacterial endocarditis causes ulceration, and with each year of growth the burden on the heart increases. Ultimately failure or bacterial endocarditis leads to death. The majority of subjects are dead at thirty years, and only seven of the 68 patients collected from the literature by Greene *et alii* were alive at fifty years. In more than half the patients cyanosis appears towards the end of the course, owing partly to a small right-to-left shunt through the *foramen ovale*, and in some patients partly to an increased oxygen consumption per unit of peripheral flow in the presence of a low cardiac output. It is not surprising to learn that the *foramen ovale* is open at autopsy in more than half the cases; during exercise in the acyanotic stage it is likely that the right auricular pressure rises and leads to auricular dilatation, which becomes more pronounced the longer these patients are watched. This would tend to keep the *foramen ovale* open, and the finding of a lowered arterial oxygen saturation with exercise supports this view. The *foramen ovale* is usually of no functional significance until the late stage of the disease, and even then, as in our cases, it may be so small as to be of no consequence.

Clinical Diagnosis.

The clinical findings have been remarkably uniform. The comparative well-being of the average patient, the absence of cyanosis and clubbing, the basal thrill and murmur, the heaving right ventricle, the diminished second pulmonary sound, the dilatation of the pulmonary artery with normal or occasionally translucent lung fields and the characteristic electrocardiogram, all make the clinical dianosis fairly secure. Particular attention must be given to the pulmonary second sound, for if it is louder than usual some other diagnosis will be the correct one. Acyanotic pulmonary stenosis does occasionally exist with patent *ductus arteriosus*, ventricular septal defect or auricular septal defect. With the first two of these associated lesions the physical, radiological and electrocardiographic findings may be indistinguishable from those of uncomplicated pulmonary stenosis. We have seen two cases in young children of patent *ductus arteriosus* associated with pulmonary stenosis in which no definite diastolic murmur was ever heard. Therefore if surgical treatment is contemplated, the diagnosis of uncomplicated pulmonary stenosis should be confirmed by cardiac catheterization. Shunting of fully saturated blood into the lungs through a patent ductus is a hindrance and not a help to these patients, particularly as it may raise the pulmonary arterial pressure and so increase the work of the right ventricle. The diagnosis of mild pulmonary stenosis when the heart size and electrocardiogram are normal is difficult. In young children particularly patency of the *ductus arteriosus* may present difficulties, as the characteristic diastolic murmur may be absent or atypical. The normal or accentuated pulmonary second sound and the character of the apex beat should enable the distinction to be made. In spite of the increase in pulmonary blood flow in this condition, the lung fields are often normal. The pulsation of the pulmonary "knob" on fluoroscopic examination may give a clue, as may widening of the pulse pressure and pulsating carotids if these are present. The *maladie de Roger* with normal-sized heart and pulmonary artery and normal lung fields cannot be distinguished with certainty from mild pulmonary stenosis without pulmonary artery dilatation. An accentuated pulmonary second sound and a thrill and murmur maximal in the third and fourth intercostal spaces would favour the former diagnosis. Commonly ventricular septal defect presents with a dilated pulmonary artery and plethoric lung fields (Wood, 1950), and in these cases the characteristic thrill sweeping from the apex towards the base combined with the right ventricular heave is quite unlike that usually found in pulmonary stenosis. Auricular septal

defects are usually large, and although the right ventricle can be felt as in pulmonary stenosis, the murmur is not nearly so impressive, and the pulmonary second sound is widely split owing to incomplete right bundle branch block (Barber, Magidson and Wood, 1950). In addition the engorgement and pulsation of the pulmonary artery and its branches make confusion with pulmonary stenosis impossible (Bedford, Papp and Parkinson, 1941). Dilatation of one branch of the pulmonary artery, usually the left, occurs occasionally in pulmonary stenosis, but pulsation, although present, is not great. If it is considerable, the presence of a left-to-right shunt should be suspected. The basal thrill and especially the murmur of aortic stenosis may be transmitted along the systemic arteries. If this sign is absent and the heart size, apex beat and electrocardiogram are normal, the diagnosis cannot be made with confidence. Theoretically such cases may occur and be confused with mild pulmonary stenosis. In the obvious case left ventricular hypertrophy should lead to the diagnosis. The pulse is usually normal in character, even when recorded graphically with a high-frequency manometer attached to an indwelling arterial needle.

Idiopathic dilatation of the pulmonary artery has much in common with mild pulmonary stenosis, and the two conditions are often confused. In fact there is no general agreement about their differentiation even on catheterization. Whether or not mild pulmonary stenosis is or is not present in these patients is a question for the future. The frequency of pulmonary systolic murmurs in hyperactive hearts which are normal makes one suspect that in some people the turbulence level is easily reached at the pulmonary valve and that idiopathic dilatation of the artery represents an adaptation to this phenomenon. One of our patients who was examined repeatedly and in whose case the diagnosis of idiopathic dilatation was made at catheterization (Figure X) lost both the thrill and the murmur after reassurance.

Treatment.

It has now been shown that surgical dilatation of the stenosed pulmonary valve is a practicable procedure (Brock, 1948; Brock and Campbell, 1950). Without surgical intervention the majority of patients with this malformation have a bad prognosis; but one is loath to recommend an intracardiac operation for every patient purely on statistical grounds. There are enough reports of patients surviving to well past middle age to make one think very carefully before recommending valvulotomy.

Three groups of patients can be distinguished, as follows. (i) Those with normal findings save for the murmur. Whatever their age they can be safely left alone, and if they are fully grown the prognosis is probably good. (ii) Those with symptoms, obviously enlarged right ventricles and auricles, ventricular strain patterns or right bundle branch block in the electrocardiogram, and grossly elevated right ventricular systolic and mean pressures. These will almost certainly need an operation to survive. (iii) The intermediate group, with few or no symptoms, only slightly enlarged or normal-sized hearts, and moderately elevated right ventricular pressures with evidence of moderate right ventricular hypertrophy or incomplete right bundle branch block in the electrocardiogram. These present the greatest difficulty. It is probable that the majority, especially the children, will ultimately pass into the second group and need operation; for the adults the prognosis is uncertain, and one can only be guided by the course of events in each case. These patients should be examined frequently, for deterioration may be rapid. Congestive failure, if treated with bed rest, digitalis, mercurials and salt restriction, is not always irreversible; but once having been present it makes anæsthesia and thoracotomy extremely hazardous.

Finally, the usual precautions against possible bacterial endocarditis should be observed in all these cases.

Summary.

1. Clinical and physiological findings in 11 cases of congenital pulmonary valvular stenosis have been presented. This lesion is not uncommon among patients with congenital heart disease and must always be considered in the differential diagnosis of acyanotic patients with basal murmurs.

2. In the moderately severe cases and the severe cases the clinical picture is sufficiently characteristic to make it recognizable by ordinary clinical methods. Mild cases in which the electrocardiogram is normal cannot be diagnosed with certainty. In both the mild and more severe cases a complicating left-to-right shunt through a patent *ductus arteriosus* or a ventricular septal defect may be unsuspected clinically and can be demonstrated only by cardiac catheterization.

3. In moderately severe cases the following signs are found: (i) A systolic murmur and thrill maximal in the second left intercostal space and propagated towards the clavicle. (ii) Right ventricular enlargement as shown by: (*a*) a palpable systolic impulse between the apex and the sternum; (*b*) forward projection of the right ventricle in the left oblique position at fluoroscopy; (*c*) an electrocardiographic pattern showing right ventricular hypertrophy or incomplete right bundle branch block in the precordial leads. (iii) Dilatation of the pulmonary artery, which may include the left main branch, with otherwise normal or diminished non-pulsatile right and left branches. The peripheral lung fields are normal.

4. In severe cases, in addition to the findings mentioned under (i) and (ii) of the third paragraph, the following are found: (*a*) a prominent "a" wave in the venous pulse, on occasion with a pulsating liver; (*b*) sometimes slight cyanosis; (*c*) dilatation of the right auricle and an enlarged pulmonary artery, with small non-pulsatile branches and abnormally translucent lung fields.

5. Severely affected patients with a small patency of the *foramen ovale* should be distinguished from those with a larger defect in the auricular septum. The latter are usually more deeply cyanosed and behave more as patients with the tetralogy of Fallot than as patients with "pure" pulmonary stenosis.

6. Statistically the prognosis is bad. It may, however, prove practicable to separate the individual patient with a good prognosis from those with a bad prognosis. Meanwhile only the latter are recommended for pulmonary valvulotomy.

Acknowledgements.

Our thanks are due to those physicians who have referred patients for study, particularly Sir Charles Blackburn, who referred the patient in Case III, and Dr. S. G. Bradfield, of the Royal Alexandra Hospital for Children, to whom we are indebted for Case VIII and others not included in this series. The remainder of the patients were referred by members of the Hallstrom Institute of Cardiology, Dr. J. K. Maddox, Dr. John Halliday, Dr. F. H. Mills and Dr. A. Seldon, who have helped us in this and other ways. Finally we owe thanks to Dr. Alan Colwell, of the X-ray department, who has provided X-ray facilities in spite of considerable inconvenience to himself.

References.

Abbott, M. E. (1936), "Atlas of Congenital Cardiac Disease", New York, American Heart Association, page 44.

Allanby, K. D., and Campbell, M. (1949), "Congenital Pulmonary Stenosis with Closed Ventricular Septum", *Guy's Hospital Reports*, Volume XCVIII, page 18.

Barber, J. M., Magidson, O., and Wood, P. (1950), "Atrial Septal Defect", *British Heart Journal*, Volume XII, page 277.

Bedford, D. E., Papp, C., and Parkinson, J. (1941), "Atrial Septal Defect", *British Heart Journal*, Volume III, page 37.

Blackford, L. M., and Parker, F. P. (1941), "Pulmonary Stenosis with Bundle Branch Block: Report of Case with Sound Tracings and Semiserial Studies of Conduction Bundle", *Archives of Internal Medicine*, Volume LXVII, page 1107.

Brock, R. C. (1948), "Pulmonary Valvulotomy for the Relief of Congenital Pulmonary Stenosis", *British Medical Journal*, Volume I, page 1121.

———, and Campbell, M. (1950), "Valvulotomy for Pulmonary Valvular Stenosis", *British Heart Journal*, Volume XII, page 377.

Burchell, H. B., and Wood, E. H. (1950), "Remarks on the Technic and Diagnostic Applications of Cardiac Catheterisation", *Proceedings of the Staff Meetings of the Mayo Clinic*, Volume XXV, page 41.

Campbell, M., and Hills, T. H. (1950), "Angiocardiography in Cyanotic Congenital Heart Disease", *British Heart Journal*, Volume XII, page 65.

Cournand, A., Baldwin, J. S., and Himmelstein, A. (1949), "Cardiac Catheterization in Congenital Heart Disease", Commonwealth Fund, New York, 1949.

Currens, J. H., Kinney, T. D., and White, P. D. (1945), "Pulmonary Stenosis with Intact Interventricular Septum", *American Heart Journal*, Volume XXX, page 491.

Dow, J. W., Levine, H. D., Elkin, M., Haynes, F. W., Hellems, H. K., Whittenberger, J. W., Ferris, B. G., Goodale, W. T., Harvey, W. P., Eppinger, E. C., and Dexter, L. (1950), "Studies of Congenital Heart Disease, IV. Uncomplicated Pulmonary Stenosis", *Circulation*, Volume I, page 267.

Durand, M., and Metianu, C. (1949), "*Présentation d'un cas de sténose congénitale pure de l'orifice pulmonaire avec vérification anatomique*", *Archives des maladies du cœur*, Number 11, page 1112.

Engle, M. E., and Taussig, H. B. (1950), "Valvular Pulmonic Stenosis with Intact Ventricular Septum and Patent Foramen Ovale", *Circulation*, Volume II, page 481.

Evans, W. (1943), "Triple Heart Rhythm", *British Heart Journal*, Volume V, page 205.

Fallot, A. (1888); quoted by Joly, F., Carlotti, J., Sicot, J.-R., and Piton, A., *loco citato*.

Greene, D. G., Baldwin, E. de F., Baldwin, J. S., Himmelstein, A., Roh, C. E., and Cournand, A. (1949), "Pure Congenital Pulmonary Stenosis and Idiopathic Congenital Dilatation of the Pulmonary Artery", *The American Journal of Medicine*, Volume VI, page 24.

Harvey, R. M., Ferrer, M. I., Cathcart, R. T., Richards, D. W., and Cournand, A. (1949), "Some Effects of Digoxin upon the Heart and Circulation in Man", *The American Journal of Medicine*, Volume VII, page 439.

Healey, R. F., Dexter, L., Elkin, M., and Sosman, M. C. (1950), "Roentgenographic Changes in Pulmonic Stenosis", *American Journal of Roentgenology and Radium Therapy*, Volume LXIII, page 813.

Joly, F., Carlotti, J., Sicot, J.-R., and Piton, A. (1950), "*Cardiopathies congénitales II: Les trilogies de Fallot*", *Archives des maladies du cœur et des vaisseaux*, Volume XLIII, page 687.

Keith, A. (1909), "The Hunterian Lectures on Malformations of the Heart", *The Lancet*, Volume II, page 359.

Lambert, E. H., and Wood, E. H. (1947), "The Use of a Resistance Wire, Strain Gage Manometer to Measure Intra-arterial Pressures", *Proceedings of the Society for Experimental Biology and Medicine*, Volume LXIV, page 186.

Myers, G. B., Klein, H. A., and Stofer, B. E. (1948), "The Electrocardiographic Diagnosis of Right Ventricular Hypertrophy", *American Heart Journal*, Volume XXXV, page 1.

Peacock, T. B. (1859), "Contraction of the Orifice of the Pulmonary Artery from Fusion of the Valves", *Transactions of the Pathological Society of London*, Volume X, page 107.

Pollack, A. A., Taylor, B. E., Odel, H. M., and Burchell, H. B. (1948), "Pulmonary Stenosis Without Septal Defect", *Proceedings of the Staff Meetings of the Mayo Clinic*, Volume XXIII, page 516.

Rappaport, M., and Sarnoff, S. J. (1949), "Electronic Multirange, Multichannel Direct Writing Pressure Recorder", *Federation Proceedings*, Volume VIII, page 130.

Selzer, A., Carnes, W., Noble, C., Higgins, W., and Holmes, R. (1949), "The Syndrome of Pulmonary Stenosis with Patent Foramen Ovale", *The American Journal of Medicine*, Volume VI, page 3.

Ungerleider, H. E., and Clark, C. P. (1939), "A Study of the Transverse Diameter of the Heart Silhouette with Prediction Table Based on the Teleroentgenogram", *American Heart Journal*, Volume XVII, page 92.

Wilson, F. N., Rosenbaum, F. F., Erlanger, H., Kossman, C. E., Hecht, H., Cotrim, N., Menezes de Oliveira, R., Scarsi, R., and Barker, P. S. (1944), "The Præcordial Electrocardiogram", *American Heart Journal*, Volume XXVII, page 19.

Wood, P. (1950), "Congenital Heart Disease", *British Heart Journal*, Volume II, page 693.

Legends to Illustrations.

FIGURE II.—X-ray films from Case I, showing a heart of normal size with "+" to "+" enlargement of the pulmonary artery, which pulsated more freely than usual. This patient is mildly affected and has almost normal exercise tolerance.

FIGURE IIIA.—X-ray films from Case II. In the antero-posterior view there is enlargement to right and left due to right auricular and right ventricular enlargement. The pulmonary artery showed "+" enlargement and on fluoroscopic examination was quiet. The branches and the lung fields were normal or doubtfully diminished.

FIGURE IIIB.—X-ray films from Case II. In the left oblique view there is apparent enlargement of both ventricles, but all the evidence suggests that only the right was enlarged. This patient was only two and a half years old, but had severe stenosis. Cardiac enlargement was present four weeks after birth.

FIGURE IV.—X-ray film from Case III. The transverse diameter of the heart is only slightly increased, but the contour is abnormal owing to "+++" enlargement of the pulmonary artery and lifting of the apex by the right ventricle. The hilar vessels and lung fields are diminished. The oblique views confirmed the presence of right ventricular hypertrophy. This patient had severe pulmonary stenosis with fairly good exercise tolerance.

FIGURE V.—Antero-posterior X-ray film of the chest in Case VIII, showing the dilatation of the pulmonary artery and its left main branch. Both pulsated noticeably.

FIGURE X.—Postero-anterior view of the chest in a case of idiopathic dilatation of the pulmonary artery. When the patient was anxious or after exercise, a systolic thrill and murmur were present in the second left intercostal space with an accentuated and split pulmonary second sound. The heart was of normal size, and at catheterisation there was no evidence of any shunt and no significant difference between the systolic pressure in the pulmonary artery and that in the right ventricle. Both were normal. After reassurance of the patient the physical signs became normal.

FIGURE XI.—Angiocardiogram in the left oblique position in Case I, taken one second after the injection of the dye. The right auricle and ventricle are normal in size, but there is slight dilatation of the pulmonary artery. Superimposed on the lower part of the superior *vena cava* and overlapping it, the pulmonary artery can be seen filling from the right ventricle. Normally the outflow tract and the pulmonary artery form an almost uniform channel; in this case the channel is interrupted by the stenosed valve, and beyond the latter the proximal part of the pulmonary artery appears to be conical, owing to the jet of dye being shot into it (one-second film). In the two-second film the proximal part of the artery had filled and appeared larger. A mild case of pulmonary stenosis with some delay in right ventricular emptying.

FIGURE XII.—Angiocardiogram taken in the left oblique view five seconds after injection of the dye in Case III. There is dilatation of the right ventricle and the pulmonary artery with delay in emptying of both. There is a constant filling defect at the pulmonary valve. The outflow tract of the right ventricle is normally formed and at catheterization was quite spacious. A severe case of pulmonary valvular stenosis.

FIGURE XIII.—Postero-anterior view of the chest of a woman, aged twenty-five years, with pulmonary valvular stenosis and patent *foramen ovale*, not included in this series. There is "+++" enlargement of the pulmonary artery and below it a greatly enlarged infundibulum. This interpretation was borne out at operation, when a successful pulmonary valvulotomy was performed. The right ventricular pressure was 114 millimetres of mercury, systolic, and four millimetres, diastolic, and that in the pulmonary artery 15 millimetres of mercury (mean). This patient also had complete right bundle branch block.

COOLEY'S ANÆMIA: A REVIEW AND PRESENTATION OF AN AFFECTED FAMILY.

By S. J. BAKER,

Registrar to the Pathology Department, Children's Hospital, Melbourne,

AND

J. J. M. O'NEILL,

Resident Medical Officer, Children's Hospital, Melbourne.

IT is probable that Cooley's anæmia is more common in Australia than is generally realized, as there are now considerable numbers of people of Mediterranean origin settled in this country. Overseas investigators have recorded figures of as high as 4% for the incidence of the minor form of the disease among communities of Mediterranean immigrants and their descendants. The object of this paper is to give a brief review of the clinical and pathological features of the disorder, to record for the first time in this country a case of the severe form of the disease in an infant, and to present a study of his available relatives. The only reports of the condition in Australian medical literature are those of Sinn (1949), who describes a boy aged nine years, and of Tebbutt (1950), who reports two adults with the disease.

The disease occurs usually in two forms, the major and the minor, although there are probably all gradations between the two (Smith, 1943).

The major form, first described by Cooley (1925, 1927, 1932), is a severe chronic, progressive anæmia occurring in childhood and characterized by the following five features.

1. Racial incidence. The majority of patients described have been people of Mediterranean origin, especially from Greece, Italy, Sicily, Syria and Armenia. More recently a small number of apparently authentic cases have been reported in other races, including Chinese (Foster, 1940), Filipinos (Silver, 1950) and Negroes (Dameshek, 1943).

2. Familial incidence. Both parents of severely affected children usually show the "minor" form of the disease. The present hypothesis is that the disease is due to a genetically determined red cell abnormality, heterozygous individuals presenting minor forms, and homozygous major forms of the disease.

3. Splenomegaly. The spleen is greatly enlarged; this is considered to be due to a combination of extramedullary hæmopoiesis and excessive blood destruction.

4. Typical hæmatological changes. The hæmatological changes result from the continued excessive destruction of the red cells, and consequent hyperplasia of the bone marrow, which releases many immature erythroid and myeloid cells into the circulation. Examination of the peripheral blood shows anæmia with great variation in the size, shape and staining reactions of the red cells, large numbers of nucleated red cells, and a leucocytosis. The diameter of the red cells varies from about 3μ to 16μ, and both small and large cells are frequent. In both stained and fresh wet preparations cells of bizarre shapes are common. Many are very thin, some give the appearance of being folded on themselves, and "target cells", with a pale rim or "halo" surrounding a darker central region, are often present. Some cells stain normally and appear to have their full complement of hæmoglobin, while others are represented merely by a thin rim of almost colourless cytoplasm. Polychromasia, basophilic stippling and a reticulocytosis of 5% to 15% are constantly present. Large numbers of circulating nucleated red cells, as many as 10,000 or more per cubic millimetre, form a striking feature of the disorder. Leucocytosis is usual, and often primitive cells, even myeloblasts, are present. The bone marrow is hyperactive and normoblastic, with an increase in the ratio of erythroid to myeloid cells. Fragility tests with hypotonic saline reveal decreased fragility, and an increased span of hæmolysis of the red cells. This is related, at least in part, to their abnormal thinness. Evidence of increased blood destruction is shown indirectly by either a raised serum bilirubin level or an increased output of urobilinogen in the fæces or urine.

5. Skeletal changes. Bone changes, the result of continuous marrow hyperplasia from early life, are detected radiologically. In the long bones the earliest described appearances are dilatation of the medullary cavity with simultaneous atrophy of cortical and of cancellous bone. These changes are followed several months later by commencing trabeculation, which, as it advances, eventually gives a characteristic mosaic pattern. This is described particularly in the metacarpals, which are often rectangular in contour. In the skull there is thickening, with widening of the diploë, followed by radial striation in parietal and frontal regions. Also reported is poor pneumatization of air sinuses. These changes in the skull appear to contribute largely to the mongoloid facies, often described in affected children (Caffey, 1937, 1951).

The minor form, which is probably about 100 times as common as the major form (Neel and Valentine, 1945), was first described in 1940 (Wintrobe), and is characteristically found in relatives of patients with the major form. The mildest cases are symptomless, while others may have symptoms referable to a mild anæmia. As with the major form, there is a familial and racial incidence. Usually there is no clinical abnormality, but in some cases the spleen is palpable.

The hæmatological changes are a minor edition of those seen in the major form. The hæmoglobin level is normal or slightly lowered, and the red cells are frequently increased in numbers, relative to the hæmoglobin value, so that a patient with a normal hæmoglobin value may have a count of 6,000,000 to 7,000,000 cells per cubic millimetre. In blood films the red cells show variations in size, shape and staining reactions, changes which are pronounced considering the hæmoglobin level, but slight compared with those of the major form. Target cells may be present, but circulating nucleated red cells are few or absent. Perhaps the most characteristic finding is decreased fragility, with an increased span of hæmolysis of the red cells in hypotonic saline.

Radiological changes in the skull and long bones are usually absent.

More severely affected subjects have clinical and hæmatological features somewhere between the typical major and minor forms. The case reported by Sinn (1949) appears to fit into this intermediate category.

Reports of Cases.

The case histories of members of a recently migrated Sicilian family exemplify the various features of the disease, a baby, aged three months, having the major form, and four adults variations of the minor form of the disease.

Case I.

J.I., a three-months-old baby, the first child of Sicilian parents, was admitted to the Children's Hospital, Melbourne, on December 26, 1950. The baby was a normal full-time infant, weighing six pounds three ounces at birth. Progress was considered satisfactory, except for mild constipation, until four days before the baby's admission to hospital, when he was feverish and vomited once. There was no family history of anæmia or jaundice; however, three of the father's siblings had died before the age of three years of unknown causes (Figure XI).

Examination of the patient showed a moderately pale, sallow baby with a distended abdomen, the weight being eleven pounds five ounces. The face and head were normal. The anterior fontanelle was small. The lymph glands in both anterior triangles and over the occiput were palpable. The abdomen was grossly distended, owing to a very large liver and spleen (Figure I). The liver edge was palpable five to six centimetres below the right costal margin, and the lower margin of the spleen was in the left iliac fossa. The only other abnormal feature was the presence of scattered petechiæ on the abdominal wall.

The following investigations were performed:

The peripheral blood was investigated on December 27, 1950. The hæmoglobin value, determined by the acid hæmatin method, was 7·2 grammes per 100 millilitres. The red cells numbered 2,600,000 per cubic millimetre. The proportion of reticulocytes was 5%. The total nucleated cell count was 38,000 per cubic millimetre, made up of white cells 28,000 per cubic millimetre and nucleated red cells 10,000 per cubic millimetre. The hæmatocrit reading was 24%, the mean corpuscular volume 92 cubic μ and the mean corpuscular hæmoglobin concentration 30%. The patient belonged to blood group A, Rh (D)-positive.

Examination of the blood film showed striking anisocytosis, poikilocytosis, polychromatophilia and stippling of the red cells. The degree of hæmoglobinization varied, some cells appearing merely as a peripheral rim of cytoplasm, others staining well throughout. The other obvious feature of the films was the large number of primitive erythroid and myeloid cells. The platelets were moderately reduced in numbers. A differential count of 200 white cells revealed the following proportions: neutrophile polymorphonuclear cells 22%, basophile polymorphonuclear cells 3%, eosinophile polymorphonuclear cells 1%, band forms 12%, metamyelocytes 3%, myelocytes 4%, premyelocytes 2%, myeloblasts 1%, lymphocytes 30%, prelymphocytes 14%, monocytes 8%. Nucleated red cells numbered 37 per 100 white cells.

These features of the peripheral blood are well seen in the accompanying Figures II, III, IV, V and VI.

The bone marrow biopsy revealed a cellular hyperactive normoblastic marrow, with an erythroid : myeloid ratio of 1:1 (the normal ratio is 1:2 to 1:3), and a shift to the left in both myeloid and erythroid series (Figure VII).

Red cell fragility was tested by a modification of Creed's technique, a photoelectric colorimeter being used to determine the degree of hæmolysis. The results are represented graphically in Figure IX, where the amount of hæmolysis occurring with each change in concentration of saline is plotted against the particular saline concentration (Bolton, 1949). The graph shows that there is an increased span of hæmolysis and an abnormally shaped curve, with a proportion of the cells showing decreased fragility.

The serum bilirubin content on December 27, 1950, was 1·4 milligrammes per 100 millilitres. The serum iron content was 0·07 milligramme per 100 millilitres. The serum protein content was 6·8 grammes per 100 millilitres, the

albumin/globulin ratio being 2:1. The serum γ globulin content was 0·63 gramme per 100 millilitres. The results of Wassermann and Kahn tests were negative. The result of a Coombs's test was negative. The maternal blood was group A, Rh (D)-positive. No agglutinating or blocking Rh antibodies were detected in the maternal serum.

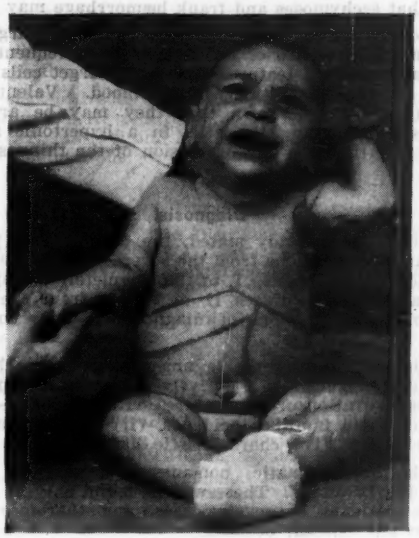

FIGURE I.

J.I. (Case I) at the age of three months, showing the degree of hepato-splenomegaly.

Radiologically the skull was within normal limits. The mandible was poorly ossified. The long bones were rather broad, with deficient modelling on relative expansion of the metaphyseal ends, thinned and "layered" cortex and rather deficient spongy bone, particularly at the wrists, knees and ankles.

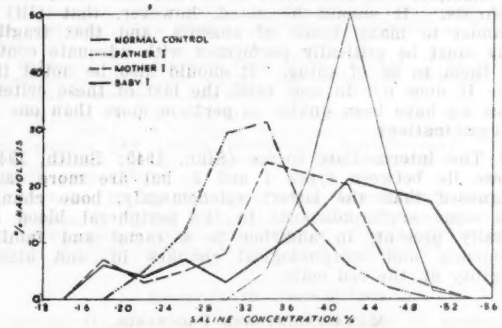

FIGURE IX.

Red cell fragility in hypotonic saline in Cases I, II and III. The degree of hæmolysis occurring with each change in concentration of saline is plotted against the saline concentration. In all three there is an increased span of hæmolysis with a proportion of cells showing decreased fragility.

Progress.

During the week following admission to hospital, the child's condition deteriorated somewhat, and the hæmoglobin reading fell to 6·0 grammes per 100 millilitres. A transfusion of 310 millilitres of compatible blood was given on January 4, 1951. After the transfusion the infant became bright, active and rowdy. The hæmoglobin value rose to 14·5 grammes per 100 millilitres, and the number of nucleated red cells diminished in the peripheral blood. The patient was discharged from hospital to his home on January 12, 1951.

Figure X shows the progressive blood findings while the child was observed in the follow-up clinic. The child put on weight rapidly, and the parents were very pleased with his progress. However, the hæmoglobin value steadily dropped, and splenomegaly and hepatomegaly persisted.

On his readmission to hospital on March 10, 1951, the child's hæmoglobin value had fallen to 5·5 grammes per 100 millilitres. He was transfused with 400 millilitres of blood and allowed to go home on the following day.

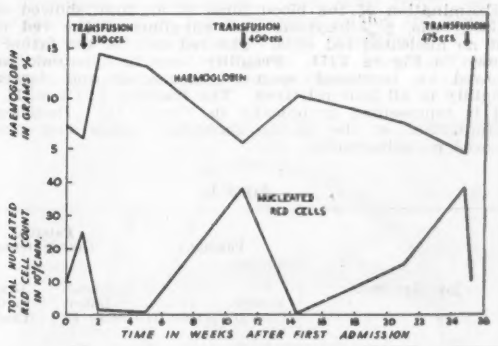

FIGURE X.

Hæmoglobin value and total nucleated red cell count in Case I. The inverse relation between the two is well seen.

The child was readmitted to hospital on May 31, 1951, very pale and extremely ill, with a hæmoglobin value of 4·3 grammes per 100 millilitres (a fall of three grammes per 100 millilitres in the space of two weeks). There were a few fresh petechiæ on the abdominal wall and upper part of the thighs, and the spleen and liver were the same as before. The findings from blood examination were as follows: the hæmoglobin value was 4·4 grammes per 100 millilitres; the red cells numbered 2,200,000 per cubic millimetre, the proportion of reticulocytes being 10%; the total nucleated

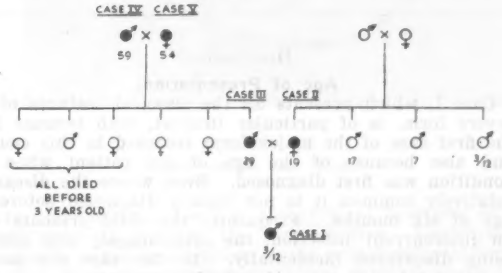

FIGURE XI.

Family tree, showing relations of J.I. (Case I). Those represented by a solid black circle were the only ones available for examination. The figures represent the ages of the respective persons. The cause of death of the father's three siblings (Case III) is unknown.

cell count was 92,700 per cubic millimetre, made up of white cells 54,700 per cubic millimetre and nucleated red cells 38,000 per cubic millimetre. The platelets were reduced in numbers, and the differential white cell count was much the same as on the first admission to hospital. Similarly the blood films and the fragility test findings showed the same features as before. Radiological examination was repeated and the following report was given: "The skull now shows some thickening of the diploë in the parietal regions, though no spicule formation or loss of outer table is yet seen. The long bones of the arm show little change, except the metacarpals which show increased trabeculation." The patient was transfused once more, with 465 millilitres of blood. His condition again improved rapidly, and he was discharged to the out-patient department.

Cases II-V.

The four relatives of J.I. available for examination (Cases II, III, IV and V) are shown in the accompanying family tree (Figure XI). These relatives exemplify the minor asymptomatic form of the disease. All four came from Sicily, and as far as could be ascertained none had had any previous illness. Clinically, no abnormality was detected in any of them, and none had splenomegaly.

Hæmatological examination of these relatives yielded the findings set out in Table I.

Examination of the blood films of all four showed some anisocytosis, poikilocytosis and stippling of the red cells, but no nucleated red cells. The red cells of the father are shown in Figure VIII. Fragility tests in hypotonic saline showed an increased span of hæmolysis and decreased fragility in all four relatives. The fragility in Cases II and III is represented graphically in Figure IX. Radiological examination of the skulls, forearms, hands and femurs showed no abnormality.

Table I.

Investigations.	Parents.		Paternal Grandparents.	
	Mother. (Case II.)	Father. (Case III.)	Grandfather. (Case IV.)	Grandmother. (Case V.)
Hæmoglobin determination (grammes per 100 millilitres)	11	13·5	16	13·8
Red cell count (millions per cubic millimetre)	6·4	7·1	6·7	7·5
Hæmatocrit reading (*per centum*)	44	46	44	42
Mean corpuscular volume (cubic μ)	69	65	66	56
Mean corpuscular hæmoglobin concentration (*per centum*)	25	29	36·5	33
Reticulocyte count (*per centum*)	<1	<1	1	<1
White cell count (per cubic millimetre)	8700 Normal.	8300 Normal.	9400 Normal.	8400 Normal.
Platelet count				
Serum bilirubin determination (milligrammes per 100 millilitres)	0·5 Negative result.	1 Negative result.	— —	0·25 Negative result.
Wassermann and Kahn tests				

Discussion.

Age of Presentation.

Case I, which presents all the classical features of the severe form, is of particular interest, both because it is the first case of the major form recorded in this country and also because of the age of the patient when the condition was first diagnosed. Even where the disease is relatively common it is not usually diagnosed before the age of six months. Frequently the child presents with an intercurrent infection, the splenomegaly and anæmia being discovered incidentally. In this case the mother was worried only about the child's constipation.

Bone Changes (Case I).

Caffey (1950) states that radiological changes are indistinct in the first year of life, and Wintrobe (1945) mentions four and a half months as the earliest age at which bone changes have been detected. It is therefore worthy of note that in Case I the patient had demonstrable changes in long bones at the age of three months, and trabeculation in the metacarpals had become quite pronounced at eight months.

Hæmatological Findings (Case I).

The changes in the blood picture occurring with varying degrees of anæmia are interesting. Most obvious is the reciprocal relationship between the hæmoglobin level and the total nucleated red cell count. This is well demonstrated in Figure X. A similar but not so pronounced relationship was noted in the character of the circulating myeloid cells: the lower the hæmoglobin value, the greater the number of more primitive types of cell. The platelets were consistently low in number, and when first examined the child had several petechiæ on the abdominal wall. The platelet count is usually recorded as being normal or high, but some cases have been recorded with reduced platelet counts (Wolman, 1946), and Wintrobe (1946) mentions that ecchymoses and frank hæmorrhage may occur.

Although the disease has been labelled "target cell" anæmia, the presence of target cells is a frequent but by no means constant feature. Moreover, target cells may be found in many conditions in childhood. Valentine and Neel (1945) have shown that they may be artificially produced by placing the cells in a hypertonic medium. Apparently they are one expression of the thinness of the cells.

Diagnosis.

Although, as already stated, the disease may probably occur in all gradations from the severe progressive anæmia of childhood, as in Case I, to the completely asymptomatic form as shown in Cases II to V, yet from the clinical viewpoint the problem of diagnosis is best considered under four headings:

1. The severe progressive anæmia of early childhood. Here the diagnosis is relatively simple and depends on the demonstration of the features outlined above—namely, (i) racial and familial incidence, (ii) splenomegaly, (iii) bone changes, (iv) characteristic hæmatological picture.

2. The asymptomatic, non-anæmic forms, as seen in Cases III, IV and V. These will be found only in relatives of the patients in severe cases, and therefore do not present any clinical problem.

3. The mild anæmia with, superficially, no obvious distinguishing features, as, for example, Case II. In these cases the diagnosis is beset with difficulties, for there are no very definite characteristics. The following criteria would appear to be necessary to establish the diagnosis: (i) a person of Mediterranean origin; (ii) a familial incidence; (iii) the presence of anisocytosis and poikilocytosis of the red cells; (iv) the presence of an increased span of hæmolysis and decreased fragility of the red cells; (v) the exclusion of all common causes of mild anæmia, for example, iron deficiency, blood loss, infections, chronic nephritis. It should be noted, however, that (iii) is common to many kinds of anæmia, and that fragility tests must be critically performed with adequate control for them to be of value. It should also be noted that Case II does not in fact fulfil the last of these criteria, since we have been unable to perform more than one set of examinations.

4. The intermediate forms (Sinn, 1949; Smith, 1943). These lie between types 1 and 3, but are more easily diagnosed than the latter; splenomegaly, bone changes and some erythroblastosis in the peripheral blood are usually present, in addition to a racial and familial incidence, and morphological changes in, and altered fragility of, the red cells.

Management and Prognosis.

As there is no known specific remedy, and as iron, liver and other therapeutic substances have no effect on the condition, the only available treatment of the anæmia is blood transfusion as necessary. Splenectomy has been performed in a number of cases, but with no essential change in the condition.

The prognosis of the disease depends upon its severity. The major form is almost invariably fatal—the earlier the condition presents itself, the shorter the course. The mild form is often compatible with a normal span of life.

Summary.

1. The incidence of Cooley's anæmia in Australia is probably much greater than is at present realized.

2. The clinical and pathological features of Cooley's anæmia are reviewed.

3. A case of the severe form of Cooley's anæmia in a three-months-old infant is recorded, together with a study of the available relatives who show features of the mild form of the disorder.

ACKNOWLEDGEMENTS.

The infant in this case was under the care of the Director of Clinical Research of the Children's Hospital, Melbourne, Dr. Howard Williams, whose help and advice we gratefully acknowledge.

We also desire to thank Dr. John Perry, pathologist to the Children's Hospital, for his advice and encouragement, and Mr. E. Matthei and Mr. C. Murphy for the preparation of the photographs.

REFERENCES.

Bolton, J. (1949), "The Distribution Curve of Erythrocyte Fragility", *Blood*, Volume IV, page 172.

Caffey, J. (1937), "Skeletal Changes in Chronic Hæmolytic Anæmias", *American Journal of Roentgenology*, Volume XXXVII, page 293.

——— (1951), "Cooley's Erythroblastic Anæmia", *ibidem*, Volume LXV, page 547.

Cooley, T. B., and Lee, P. (1927), "A Series of Cases of Splenomegaly in Children with Anæmia and Peculiar Bone Changes", *Transactions of the American Pediatric Society*, Volume XXXVII, page 29 (also published in *American Journal of Diseases of Children*, Volume XXX, 1925, page 447).

Cooley, T. B. (1932), "Erythroblastic Anæmia", *American Journal of Diseases of Children*, Volume XLIII, page 705.

Dameshek, W. (1943), "Familial Mediterranean Target Oval Cell Syndromes", *The American Journal of the Medical Sciences*, Volume CCV, page 643.

Foster, L. P. (1940), "Cooley's Syndrome in a Chinese Child", *American Journal of Diseases of Children*, Volume LIX, page 828.

Neel, J. V., and Valentine, W. N. (1945), "The Frequency of Thalassemia", *The American Journal of the Medical Sciences*, Volume CCIX, page 568.

Silver, H. K. (1950), "Mediterranean Anæmia in Non-Mediterranean Children", *American Journal of Diseases of Children*, Volume LXXX, page 767.

Sinn, H. J. (1949), "Cooley's Anæmia: Report of a Case", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 579.

Smith, C. H. (1943), "Familial Blood Studies in Cases of Mediterranean Anæmia", *American Journal of Diseases of Children*, Volume LXV, page 681.

Tebbutt, A. H. (1950), "Mediterranean (Cooley's) Anæmia in Australia", THE MEDICAL JOURNAL OF AUSTRALIA, Volume I, page 50.

Valentine, W. N., and Neel, J. V. (1945), "The Artificial Production of Target Cells", *The American Journal of the Medical Sciences*, Volume CCIX, page 741.

Wintrobe, M. M., Mathews, E., Pollack, R., and Dobyns, B. M. (1940), "A Familial Hæmopoietic Disorder in Italian Adolescents and Adults, Resembling Mediterranean Disease", *The Journal of the American Medical Association*, Volume CXIV, page 1530.

Wintrobe, M. M. (1946), "Clinical Hæmatology", page 565.

Wolman, J. J., and Dickstein, B. (1946), "Changing Concepts in Mediterranean (Cooley's) Anæmia", *The American Journal of the Medical Sciences*, Volume CCXII, page 722.

Legends to Illustrations.

FIGURE II.—Peripheral blood of J.I. (Case I), showing the variations in size, shape and staining of the red cells and four nucleated red cells. Leishman stain; × 650.

FIGURE III.—Peripheral blood of J.I. (Case I), illustrating the abnormalities of the red cells. Leishman stain; × 1250.

FIGURE IV.—Peripheral blood of J.I. (Case I), showing a myelocyte, together with three lymphocytes, a polymorphonuclear leucocyte and two nucleated red cells. Leishman stain; × 1250.

FIGURE V.—Peripheral blood of J.I. (Case I), showing a myeloblast with well-defined nucleoli, and four nucleated red cells. Leishman stain; × 1250.

FIGURE VI.—Peripheral blood of J.I. (Case I), showing four primitive nucleated red cells. Leishman stain; × 1250.

FIGURE VII.—Bone marrow smear from J.I. (Case I), showing active normoblastic hyperplasia. Leishman stain; × 650.

FIGURE VIII.—Peripheral blood of the father of J.I. (Case III). This illustrates the variation in size, shape and staining of the red cells as seen in all four relatives of J.I. Note the stippled cell and the "target cell". Leishman stain; × 1250.

AIDS TO EASY BREATHING.[1]

By DOUGLAS G. RENTON, F.F.A.R.C.S.,

Anæsthetist, Neuro-surgical Unit, Alfred Hospital, Melbourne.

Of the various factors in the ætiology of post-anæsthetic atelectasis and pulmonary œdema, the one which plays the greatest single part and which is almost invariably present is impedence to inspiration. This same factor has considerable bearing on the pulmonary œdema and atelectasis which develop independently of anæsthesia in many medical and surgical conditions, such as coma from any cause and asthenia from severe illness.

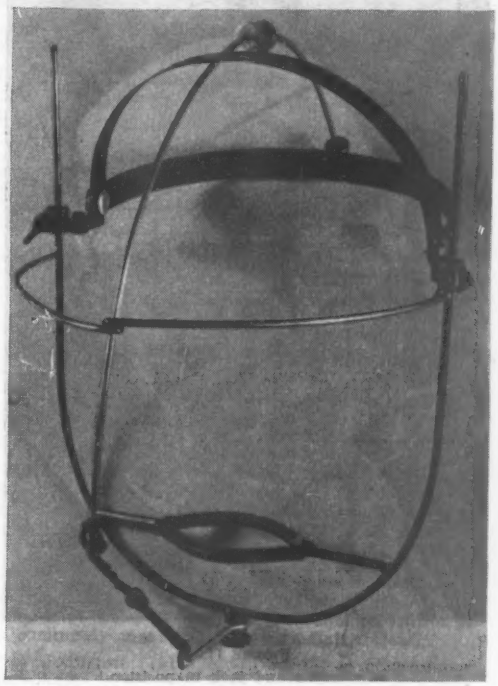

FIGURE I.
Jaw cradle and jack.

During anæsthesia, one of the cardinal functions of the anæsthetist is to keep the airways free. Unobstructed breathing is just as important in the post-anæsthetic period. A tour through general medical and surgical wards will frequently reveal a number of patients, who have not necessarily been anæsthetized, suffering from laboured breathing. They are in urgent need of relief but are usually receiving little. The *placebo* for laboured breathing is the nasal catheter and a trickle of oxygen, and this is often the only help being given.

How many patients really need to be given oxygen? All need air—plenty of air without having to fight for it. What is needed is a lightening of the load on their breathing. If this load can be lightened there will be little need for oxygen and the incidence of pulmonary œdema will be reduced. If œdema is already present the lightening of the load will remove one causal factor and improve the prospects of recovery.

The point may be illustrated specifically by cases of head injury. Those victims who die invariably are found to

[1] Read at a meeting of the Australian Society of Anæsthetists on February 23, 1951, at Sydney.

have pulmonary œdema at autopsy. There is experimental evidence (Cameron and De, 1949) to suggest pulmonary œdema of purely neurogenic origin, but this is most probably a catastrophic œdema with early death. The pulmonary œdema which develops during the later course of head injuries is mainly the result of respiratory impedence, particularly of inspiratory impedence.

Many patients with head injuries must die because of the extent of their cerebral damage. Others will recover with surgical treatment. Yet others, who might be brought successfully to operation, are allowed to die from untreated pulmonary œdema. Such œdema can often be prevented by

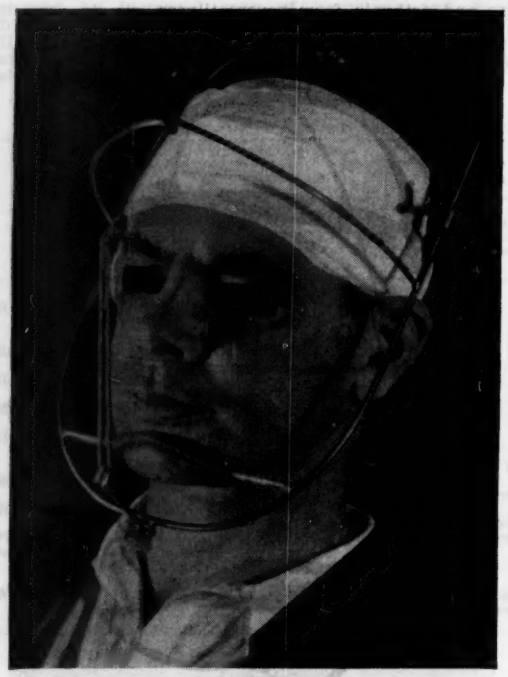

FIGURE II.
Jaw cradle and jack in position.

unremitting care of the airways, and this should take first priority in the treatment of these patients. In the management of cerebral compression—for example, in middle meningeal hæmorrhage—the maintenance of a free airway, by tracheotomy if necessary, and of adequate lung ventilation, if necessary by mechanical means, is of importance equal to the surgical relief of the compression. Neither is of value without the other.

The Oro-Pharynx and the External Nares.

There are two vulnerable regions in the air passages where resistance to breathing most frequently originates —the oro-pharynx and the external nares.

The Oro-Pharynx.

The front wall of the oro-pharynx is formed by the mobile base of the tongue and the attached epiglottis. Muscular tone keeps the pharynx patent. With decrease or loss of muscular tone, the tongue and epiglottis fall back under the influence of gravity, occlude the lumen to a greater or less extent and impose a load on breathing. In the unconscious or anæsthetized patient, in whom the sensory side of the respiratory reflex arc is abolished, a suitable oral airway, suitably placed, will overcome the obstruction. An improperly placed airway may lift forward the base of the tongue and at the same time jamb the epiglottis down over the glottis, converting the epiglottis into a "no-way valve", obstructing both inspiration and expiration.

Patients in whom the sensory side of the reflex arc is more or less active will not tolerate an oral airway. Some other means must be adopted to lift the base of the tongue clear of the posterior pharyngeal wall. This may be done manually by supporting the lower jaw. A finger under the symphysis will often be sufficient, the apposing of the

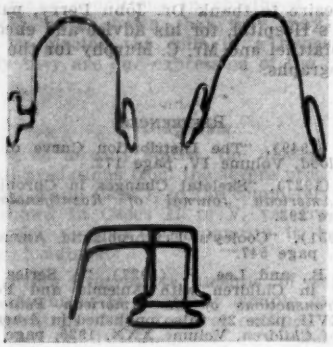

FIGURE III.
Self-retaining nasal retractors.

teeth giving sufficient lift to the base of the tongue. In the edentulous, such a lift brings the alveolar margins into apposition, reducing the capacity of the mouth and rendering it too small to accommodate the tongue, which must therefore find accommodation in the pharynx and there restrict the airway. A forward lift to the jaw by

FIGURE IV.
Nasal retractor in position.

pressure behind one or both angles of the mandible is needed. It is not unusual for the pursed lips of the edentulous to occlude the nares partially or completely. The lips must be held apart either by the fingers or by some form of bite block.

This reduction in the capacity of the mouth in the edentulous provides the main reason for leaving full dentures in position during the induction of anæsthesia. They are left in to keep the airway patent and to act as a prophylactic against the development of spasm, not removed for fear of spasm.

Manual support of the jaw necessitates the continuous presence of an attendant, or a relay of attendants, which may be difficult to arrange in a busy ward. Although there can be no relaxing of supervision, methods are available to replace manual support of the jaw and free the attendant for other duties in close proximity to the patient. A loop of bandage passed under the chin and tied to the head of the bed will often give the necessary support. A knot in the bandage under the notch of the symphysis will prevent the loop from slipping forwards over the chin and retain it in position. On odd occasions a suture has been passed through the skin under the symphysis and the jaw suspended from some overhead support.

Some patients require support for a considerable time. For these, the apparatus illustrated in Figures I and II was devised. It is an adjustable jaw cradle and jack. The cradle lifts the jaw cranially, and this upward lift may be sufficient to clear the pharyngeal airway. More often, a forward lift also is required. This forward lift is obtained by means of a jury mast fixed to the retaining head bands, projecting forwards in front of the face and ending in a hook some two to four inches in front of the tip of the nose. An elastic band from the hook to the cradle gives the desired forward lift. The band does not prevent voluntary movement of the jaw in conscious patients, but does encourage a return of the jaw to the forward position without conscious effort.

The External Nares.

In the architecture of the air passages, the smallest cross-sectional area is at the external nares. The *alæ nasi* are mobile, but their movement is passive. During inspiration, especially if it is laboured, the alæ are sucked inwards and so further narrow the narrowest part of the airway, thus adding to the load on inspiration.

In the unconscious or anæsthetized patient, the resistance to breathing offered by the *alæ nasi* can be partially overcome by the use of a rubber nasal airway—that is, a half-length Magill catheter—which reaches down into the pharynx behind the base of the tongue and gives a free airway to just above the glottis.

The passing of such a tube may be a life-saving measure in the management of the respiratory spasm sometimes encountered during the induction of anæsthesia. Of patients who develop spasm, nearly all have some degree of resistance to inspiration prior to the development of the spasm, and this most frequently at the external nares. The majority of "spasm-prone" patients can be recognized within a few moments of the commencement of the administration. As soon as mouth breathing ceases, any resistance to breathing through the external nares can be detected immediately and steps taken to lighten the inspiratory burden.

The Treatment of Spasm.

In the treatment of an established spasm, the first step is the passage of a nasal tube. When air is thus made more readily available to the patient, the larynx will immediately relax and accept it, and as soon as air enters the lungs without undue effort, the spasm of the muscles of respiration, intrinsic and accessory, will pass off. The jaw will then relax, allowing the easy placing of a pharyngeal airway with a much larger bore than any possible nasal tube.

The conscious or semi-conscious patient will tolerate neither a nasal nor a pharyngeal airway. Any help offered to such a patient must therefore produce no irritation or annoyance. Figures III and IV illustrate a self-retaining nasal retractor, which holds open the external nares, prevents sucking-in of the *alæ nasi* during inspiration and so frees the breathing of this resistance. It was made of light stainless steel wire in several sizes by Mr. E. Dixon, the instrument maker at the Alfred Hospital. It is sound practice to test appliances on oneself. This was done. After a few minutes' strangeness one is gratefully aware of free and easy breathing. There is no irritation or discomfort. In fact, I now wear such a retractor to bed almost every night.

During induction of anæsthesia, as soon as it appears that a patient is "spasm-prone" the mask is removed and a retractor inserted. With the lightening of the load on breathing, the feeling of suffocation is relieved, the induction proceeds smoothly, and spasm rarely develops.

The retractor offers a measure of assistance to many types of dyspnœic patients in whom there are a narrowing of the nares and a sucking-in of the *alæ nasi*.

Summary.

Impedence to respiration, particularly to inspiration, is a most potent factor in the ætiology of pulmonary œdema and atelectasis.

The most common sites for respiratory impedence are (i) in the oro-pharynx and (ii) at the external nares.

A self-retaining jaw cradle and jack and a self-retaining nasal retractor, both aids to easier breathing, are illustrated.

Reference.

Cameron, G. R., and De, S. N. (1949), "Experimental Pulmonary Œdema of Nervous Origin", *The Journal of Pathology and Bacteriology*, Volume LXI, page 375.

DIPHYLLOBOTHRIUM LATUM (LINNÉ) IN AUSTRALIA.

By Dorothea F. Sandars,
Queensland Institute of Medical Research, Brisbane.

The following appear to be the only Australian records of *Diphyllobothrium latum*.

In 1906 a specimen of *Diphyllobothrium* was recovered by Elkington (1908) from a Syrian in Tasmania and described by Stephens (1908) as *Diphyllobothrium parvum*. Johnston and Cleland (1937) state that Zschokke has shown this to be a synonym of *Diphyllobothrium latum*.

T. Harvey Johnston (1909) exhibited a specimen of *Dibothriocephalus latus* from man in New South Wales. He noted that it was "the only Australian record of a tapeworm commonly met with near the Baltic Sea".

T. Harvey Johnston (1911) again exhibited a specimen of *Bothriocephalus latus* from New South Wales. It seems probable that this specimen may have been the one previously exhibited (1909).

Sweet (1924) noted that the two infections with *Dibothriocephalus latus* reported during the hookworm campaign "were found in persons resident in Queensland, who had come from Finland".

Cilento, McIntosh and Charlton (1924) noted the occurrence of *Diphyllobothrium latum* in two Finns at Nambour (Queensland) and in a Swiss at Cairns. The former are probably the same as the records by Sweet (1924) and Charlton (1924). A doubtful case was also reported from the Newcastle-Maitland area.

Penfold *et alii* (1936) recorded two infections from Finns in Victoria.

Gordon (1940) recorded *Diphyllobothrium latum* from two greyhound dogs from Picton Lakes, New South Wales, and from one greyhound dog from Kempsey, New South Wales.

In March, 1951, several proglottides from a patient from the Brisbane General Hospital were identified as *Diphyllobothrium latum*.

The patient, a young man, aged eighteen years, was an immigrant from Ireland, who had visited England. Sixteen months previously he had known that he had a tapeworm, and at intervals since he was, by personal observation, aware of proglottides in his stools. During this time he had lost weight. On treatment with filix mas (in March, 1951) a great length of worm was reputed to have been passed.

The writer unfortunately had the opportunity to examine only a few of the proglottides, which had already been preserved. These were of the correct size, they had the characteristic rosetted convolutions of the uteri in the mid-part of the proglottides, and the eggs *in utero* were within the size range for *Diphyllobothrium latum*.

At the present time there is a much greater chance than normally that this tapeworm may be introduced into Australia, because of the influx of "New Australians". Many of these immigrants come from endemic areas for this worm. There can be little doubt that many Australian animals, invertebrate and vertebrate, would serve both as intermediate hosts necessary for the developmental stages of this tapeworm, and also as "reservoir" hosts for the sparganum larval stage infective to man. This being the case, it is important to realize that this tapeworm may readily become endemic in Australia.

In the life history of *Diphyllobothrium latum*, the ovoidal, operculate eggs pass out with the fæces of the host, and reaching water, hatch to produce ciliated larval forms (coracidia), which swim around freely and invade a water-flea (Copepoda). Water-fleas are abundant in Australia. *Diaptomus graciloides*, which serves as an intermediate host for *Diphyllobothrium latum*, has been recorded in New South Wales (Playfair, 1914); two other species of *Diaptomus* have been recorded from New South Wales and Queensland (Henry, 1923). In this host the parasite develops into the procercoid larval form. If an infected water-flea is then consumed by some suitable fish (it is possible that other animals, such as frogs, may serve as "reservoir" hosts), the procercoid invades the muscular tissues and becomes a plerocercoid or sparganum, which is the stage infective to the final host.

It seems probable that any of the numerous fresh-water fishes of Australia would be suitable hosts for the spargana of *Diphyllobothrium latum*; trout, which have been introduced into many Australian streams, have been recorded as hosts in Europe. Man is infected by the consumption of raw or undercooked fish infected with spargana. When spargana lie between the muscles towards the middle of the body of the intermediate host, they may escape death, even though the fish would be considered to be "cooked".

It is suggested that, because of the importance to Australia of this matter, medical practitioners who diagnose and recover tapeworms from patients should make every effort to have these parasites correctly identified. The writer would willingly cooperate in identifying any material forwarded, and would appreciate receiving either fresh or preserved specimens. Fresh stools from infected patients would also be of use for experimental work.

Preservation of Tapeworms.

The specimen should be preserved in either 10% neutral (buffered) formalin solution or in alcohol (70%).

To facilitate specific identification, several proglottides should be removed and flattened (for example, between two pieces of glass—additional pressure may be necessary). They must then be fixed by the introduction of 10% formalin solution between the flattening surfaces and left for approximately fifteen minutes. These proglottides may then be preserved in either formalin (10%) or alcohol (70%).

Addendum.

Further specimens of Diphyllobothrium, collected from hosts from the Brisbane area, have since been examined. This material was kindly loaned by Mr. P. J. O'Sullivan, of the Animal Health Station, Brisbane. It includes the following: (*a*) a portion of *Diphyllobothrium latum* from a migrant from either Russia or the United States of America, collected in 1930, presumably in Brisbane; (*b*) a considerable length of Diphyllobothrium from a dog in Brisbane, collected in 1939. This appears to be *Diphyllobothrium latum*; (*c*) two specimens of *Diphyllobothrium latum* from two dogs in Brisbane, taken respectively in June and October, 1946.

References.

Cilento, R. W., McIntosh, R. D., and Charlton, N. B. (1924), "The Diagnosis of Bowel Diseases in Northern Australia", Commonwealth of Australia, Department of Health Service Publication (Tropical Division), Number 5, page 80.

Charlton, N. B. (1923), "Bowel Parasites of Australia and its Dependencies. Their Relation to Disease and their Frequency of Occurrence", "Transactions of the Australasian Medical Congress (British Medical Association)", First Session, page 324.

Elkington, J. S. C. (1908), "Notes on a New Human Cestode (Dibothriocephalus Parvus)", "Transactions of the Australasian Medical Congress", Eighth Session, Number 2, page 337.

Gordon, H. McL. (1940), "The Occurrence of the Broad Fish Tapeworm of Man and Carnivores in Dogs in Australia", The Medical Journal of Australia, Volume I, page 47.

Henry, M. (1922), "A Monograph of the Fresh-Water Entomostraca of New South Wales, Part II. Copepoda", *Proceedings of the Linnean Society of New South Wales*, Volume XLVII, page 552.

Johnston, T. Harvey (1909), "Notes and Exhibits of Entozoa", *Proceedings of the Linnean Society of New South Wales*, Volume XXXIV, page 117.

——— (1911), "Exhibit of Specimens of Animal Parasites", *The Australasian Medical Gazette*, Volume XXX, page 336.

———, and Cleland, J. B. (1937), "A Survey of the Literature Relating to the Occurrence in Australia of Helminth Parasites of Man", *Transactions of the Royal Society of South Australia*, Volume LXI, page 255.

Penfold, W. J., and Penfold, H. B. (1936), "The Diagnosis of Tænia Saginata Infestation", The Medical Journal of Australia, Volume I, page 317.

———, and Phillips, M. (1936), "A Survey of the Incidence of Tænia Saginata Infestation in the Population of the State of Victoria from January, 1934, to July, 1935", The Medical Journal of Australia, Volume I, page 284.

Playfair, G. I. (1914), "Contributions to the Knowledge of Biology of the Richmond River", *Proceedings of the Linnean Society of New South Wales*, Volume XXXIX, page 93.

Stephens, J. W. W. (1908), "Two New Cestodes and a New Lingulatulid", *Annals of Tropical Medicine and Parasitology*, Volume I, page 549.

Sweet, W. C. (1924), "The Intestinal Parasites of Man in Australia and its Dependencies as Found by the Australian Hookworm Campaign", The Medical Journal of Australia, Volume I, page 407.

Reports of Cases.

A DANGEROUS OPERATION.

By D. F. Lawson, M.R.C.O.G.,
Women's Hospital, Melbourne.

Dilatation of the cervix and curettage of the uterus is a dangerous operation. It can be the means of tearing the cervix, of introducing infection, of rupturing a pyometra, and on occasions of producing intestinal obstruction. Bowel has been pulled into a uterus by sponge forceps groping blindly through a perforation. Without necessarily believing it, we can repeat the pathologist's story of his report on curette scrapings: "The tissue presented for examination strongly resembles intervertebral disk tissue."

This dangerous operation is perhaps most dangerous when it is performed in the first few weeks *post partum*. It is dangerous because of the adverse effect it can have upon the capacity of the uterus to function in a normal way.

These brief notes on patients, all of whom I have treated in the short space of twelve months, will, I hope, substantiate these fears we have about the operation.

Case I.

Mrs. M., aged thirty-six years, had two children, aged nine years and four years respectively. A month after the birth of the second child her uterus was curetted on account of heavy bleeding *per vaginam*. From that time her menstrual flow lasted for only one day instead of for four to five days, as had been her previous pattern, and she had a yellow intermenstrual discharge. The patient wanted to have a third child and consulted me to see that all was well with her before she embarked on a pregnancy. The relevant findings on examination were that there was an extensive vaginal erosion and that I was unable to pass a sound into the uterus, which was normal in size, shape and position.

At the patient's second visit three months later I was still unable to pass a sound into the uterus, so advised the patient to be admitted to hospital. Under anæsthesia a sound was eventually forced through the stenosed cervical canal, and the rigid canal was with difficulty dilated to admit a number 8 Hegar's dilator. A number 8 rubber catheter was then stitched into the cervical canal and left *in situ* for five days, during which time the patient was given sulphonamides and penicillin prophylactically.

Over the next three months I examined the patient at regular intervals and passed closed uterine packing forceps into the uterine cavity, hoping in that way to maintain the patency of the cervical canal. When last I examined the patient it seemed that the patency of the cervical canal was established, the menstrual flow was lasting two to three days, and the cervical erosion, which I had been touching with pure carbolic acid at each visit, was healed.

Comment.

I regarded this patient as having cervical stenosis and endometrial destruction following post-partum curettage.

Case II.

Mrs. A.C. had been married for eleven years and had had a myomectomy performed before she became pregnant for the first time. After the labour, which was terminated by a difficult mid-forceps delivery, she developed a severe puerperal infection which responded slowly to treatment. When the child was six weeks old the patient had a fairly severe uterine hæmorrhage. Examination revealed a patulous cervical canal with what appeared to be a piece of placental tissue in the process of being extruded. At curettage I found this "placental tissue" very tough and adherent. None of the tissue removed was sent for pathological examination, but in retrospect I think that what I had taken to be placental tissue was in fact a small fibroid tumour in the process of being extruded.

The patient reported next fifteen months later, having had no menstrual periods since the delivery of her child. With difficulty a sound was passed into the uterine cavity. The diagnosis of cervical stenosis was made and the patient was admitted to hospital. The cervical canal was with difficulty dilated to admit a number 8 Hegar's dilator and a number 8 rubber catheter was sewn into the cervical canal and left *in situ* for seventy-two hours. Routine chemotherapy was given post-operatively. Curettage was performed on this patient, and the pathologist's report read: "Uterine muscle seen but no endometrium." Although this patient had not menstruated she had regular menstrual molimina and I assumed that the ovarian function was normal. A basal temperature chart was not made to confirm this suspicion.

Comment.

In this case I think that the amenorrhœa is due to the fact that in a too vigorous curettage I deprived the uterus of all its endometrium; perhaps I completed a destruction commenced by the infective process.

Case III.

Mrs. N., aged thirty years, had two children, one aged seven years and one aged six months. After the birth of the second child her uterus was curetted twice at intervals of four days when the child was four weeks old. Bleeding had been free and she was given a transfusion of five pints of blood. There had been no menstruation since the confinement. The child had been artificially fed from the age of four weeks.

On examination of the patient it was not possible to pass a sound into the cavity of the uterus. At operation on June 29, 1950, a sound was eventually pushed into the uterus, the cervical canal was dilated and a number 10 catheter was sewn in and left for seventy-two hours. Packing forceps were subsequently passed into the uterine cavity at regular intervals, and on August 2, when the child was approximately ten months old, the patient menstruated for the first time; she has continued to do so at regular intervals. The flow is scanty and lasts for three or four days. Previously the patient had had what she regarded as a normal flow lasting for four or five days.

The diagnosis made was cervical stenosis and endometrial destruction.

Case IV.

Mrs. W., aged twenty-seven years, had one child aged two and a half years, and twins aged ten and a half months. When the twins were six weeks old her uterus was curetted because of hæmorrhage. From the time when the twins were five months old the patient had had regular attacks of lower abdominal discomfort lasting twenty-four hours. She described the discomfort as being like that which she had had with her menstrual periods. The most recent attack had been more severe than her previous ones, and the discomfort had lasted for three days. The first attempt to pass a sound into the uterine cavity was not successful, but after a little more force had been used the sound went into the cavity and dark altered blood began to issue forth; the diagnosis of cervical stenosis with hæmatometra was established. At operation a Duke's stem was sewn into the cervical canal and left for five days, and this was followed with regular dilatation. The patient now menstruates normally.

Case V.

Mrs. G.H., aged twenty-nine years, had had a normal confinement for her first child. The second confinement was normal too, but on the eighth day *post partum* free hæmorrhage occurred, so I curetted her uterus—very gently I thought. All that was obtained at curettage was dark old blood clot with no recognizable products of conception.

Menstruation commenced when the child was nine months old, but was very scanty, the whole process being completed in twenty-four hours. Previously menstruation had lasted three or four days. This patient's third child was delivered for me, during my absence on holidays, by Professor Lance Townsend, who had to remove manually a very densely adherent placenta.

Comment.

In this case I consider that my "gentle" curettage damaged the endometrium sufficiently to make menstruation scanty and placentation in the subsequent pregnancy abnormal.

Case VI.

Mrs. M., aged thirty-two years, had had a Cæsarean section for her first child because of a transverse lie. The second and third confinements were normal. A month after the third confinement her uterus was curetted because of bleeding.

I examined her when her child was twelve months old, and up till that time she had had no menstrual periods since her confinement. I was unable to pass a sound into the uterine cavity. Without any treatment this patient subsequently had two scanty menstrual periods and then became pregnant again. Cæsarean section was performed at the thirty-seventh week because of a central *placenta prævia*.

Comment.

It is possible that the sequence of events was as follows: endometrial damage by curettage, delayed onset of scanty menstrual periods, abnormal placentation, *placenta prævia*.

Case VII.

Mrs. M. was aged thirty-one years. Her first child was stillborn, and a month after her confinement her uterus was curetted because of hæmorrhage.

I examined her in consultation two hours after the birth of her second child. The placenta was retained and there was no bleeding. I attempted to remove the placenta manually. It was densely adherent and I felt that I had been able to remove about half of it. Fear of perforating the uterus with my fingers made me desist. The patient

was subsequently given sufficient blood to bring her hæmoglobin value up to 90% (of 14 grammes) and routine chemotherapy, and her puerperium was fortunately uneventful.

Comment.

In this instance the sequence of events was as follows: curettage, endometrial damage, abnormal placentation and *placenta accreta*.

Discussion.

The clinical histories of seven patients who had had post-partum curettage are described. In two cases the curettage was performed by the resident staff at the Women's Hospital, in three cases the curettage was performed by general practitioners, and in two cases I performed the post-partum curettage myself.

It would seem that in performing this operation it is very easy to remove the whole thickness of the endometrium and leave scarred areas in the uterus. It would also seem that the cervical canal is often curetted rather deeply, and that as a result of this cervical stenosis, and on occasions cervical adhesions and occlusion of the canal, can be produced.

When one is performing curettage *post partum* it would seem highly desirable to use a blunt curette very gently and to be very careful to avoid curettage of and damage to the cervical canal.

Reviews.

UNIPOLAR ELECTROCARDIOGRAPHY.

To many physicians of an earlier maturity who began their study of electrocardiography with the empirical work of Katz and who sorrowfully saw their hopes of conquering the new unipolar technique fail because of inadequate literature, the appearance of a new work on unipolar electrocardiography will be warmly welcomed, especially since this new book of some 200 pages is an example in clarity of expression and illustration.[1]

This fine book covers the physiological principles of depolarization and repolarization in a manner which leaves no doubt that these fundamentals must be grasped to give a broader ability to interpret unipolar tracings and that certain set patterns must not be learned parrot-wise.

Fundamental heart positions are discussed; these are based on Wilson's six positions. Anatomically the discussion seems like splitting hairs, but it does explain certain patterns in (*a VL*) and (*a VF*) leads which would otherwise remain a mystery—for example, when there is a (*qR*) pattern in both these leads.

Hypertrophy patterns are given in the light of the most modern concepts, and this section of the book is followed by a clear discussion of bundle branch blocks.

The chapter on myocardial infarction is well done and the physiology carefully explained; eight sites of localization are given and are worth memorizing.

Infarction complicating bundle branch block is mentioned and clues are given for its detection in the presence of left bundle branch block.

It is interesting to note that Sokolow is quoted as saying that 5% of normals show an *rSR* pattern in lead V_1. This is important to remember, as some patients with a systolic pulmonary murmur and such a pattern in the electrocardiogram in V_1 may wrongly be considered as suffering from patent interatrial septal defects in view of the frequency of right bundle branch block in these cases.

The illustrative tracings at the end of the book are of the same high standard as the rest. Figure 109, page 155, is interesting as it is a normal tracing which shows inversion of *T* waves right across the precordium from V_1 to V_6 due to the presence of marked clockwise rotation.

Figure 171, page 200, is contentious as it purports to show acute pericarditis with elevated *ST* segments. These are not elevated if they are referred to the *TP* segments as a reference level as recommended on page 144.

All in all, this new work must become a standard handbook on unipolar electrocardiography.

[1] "Clinical Unipolar Electrocardiography", by Bernard S. Lipman, A.B., M.D., and Edward Massie, A.B., M.D., F.A.C.P.; 1951. Chicago: The Year Book Publishers, Incorporated. 9" × 6", pp. 232, with 191 illustrations. Price: $5.00.

PRIMER ON FRACTURES.

A "Primer on Fractures" has been prepared by the Special Exhibit Committee of Fractures in cooperation with the Committee on Scientific Exhibits of the American Medical Association.[1] Kellogg Speed in a foreword states that the book is intended for ready-at-hand use by students and practitioners.

This small work contains much useful information for students and also some elementary advice; it is, however, so brief that on many aspects it is nude. It consists of 53 printed pages only, including illustrations. Blank pages alternate with printed pages and are to be used for notes. It is to be hoped that the blank page opposite the fracture of a metacarpal bone will be full of notes dictated by irate teachers condemning in vivid language the illustrated method of treatment of fracture of a metacarpal bone. In some parts of the world the use of a roller bandage in the palm as a splint to immobilize a fracture of a metacarpal bone would be regarded as culpable negligence. This is the most serious error. The use of a banjo splint in the treatment of a fracture of a phalanx cannot be regarded with favour. The use of continuous pin traction through the calcaneus for the treatment of a fracture of the femur or of a fracture of the tibia and fibula should be condemned.

The emphasis on conservative treatment is wise teaching for students. Nevertheless it is strange to read these days that "it is also possible to use internal fixation to fasten a fractured hip".

Most of the illustrations, which are drawings, are good; some are futile. Whilst there are many good features in this book, because of certain errors we cannot recommend it to students or practitioners.

THE DESIGN OF EXPERIMENTS.

R. A. Fisher's "The Design of Experiments" was first published in 1935 to amplify remarks made in his "Statistical Methods" on the necessary relation between the design and the interpretation of experiments. These two books have revolutionized quantitative biological research. "The Design of Experiment" is now in its sixth edition, testifying to its popularity.[2] It begins with a brief discussion of induction, the argument from the particular to the general, by which we may hope to add to our knowledge by experimentation. The principles of experimentation are exemplified by a simple experiment, in which a lady's assertion that she can tell the order of addition of the ingredients of a cup of tea by tasting is tested, and by the discussion of an experiment in cross fertilization by Charles Darwin. Fisher's early work had been done at the agricultural station at Rothamsted, and thus many of the examples in the following chapters have been drawn from agriculture. This does not limit the field of application of the methods, however, for they have been successfully applied to many other fields, such as experimental pharmacology and the analysis of laboratory errors. A very common statistical technique, whose value is well displayed in the book, is the analysis of variance, originally developed by Fisher. Proper design of experiment ensures that this technique has its greatest effectiveness in dealing with the particular problem. A point of importance is that many different comparisons can be made simultaneously and their interactions determined. This is more economical in material than the more classical method of allowing the factors to be tested to vary one at a time. Here again, while the examples are agricultural, the applications to other sciences, such as experimental medicine, are apparent.

This book is the classic in the field and can be recommended to all interested in experiments of a numerical nature.

[1] "Primer on Fractures", prepared by the Special Exhibit Committee on Fractures in cooperation with the Committee on Scientific Exhibit of the American Medical Association; Sixth Edition; 1951. New York: Paul B. Hoeber, Inc. 9¼" × 6", pp. 121, with illustrations. Price: $2.00.

[2] "The Design of Experiments", by Ronald A. Fisher, Sc.D., F.R.S.; Sixth Edition; 1951. Edinburgh: Oliver and Boyd. 9" × 6", pp. 260, with five figures. Price: 12s. 6d.

The Medical Journal of Australia

SATURDAY, OCTOBER 20, 1951.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

MENTAL HYGIENE.

ALTHOUGH passing reference has been made from time to time to mental hygiene and attention has often been drawn to it by implication, it is nearly twenty years since a formal discussion of the subject was published in these columns. The Victorian Council for Mental Hygiene had issued its second annual report, and this report was discussed. It was pointed out on that occasion that the original aim of the mental hygiene movement was the alleviation of the suffering and the better treatment of the mentally afflicted. Its present-day aims were stated to be very much wider. "Mental hygienists not only seek to mitigate the suffering of those afflicted with the major psychoses, but to conserve the mental health of the whole community by preventing, as far as possible, all kinds of nervous and mental disorder or defect. They strive for the better care and training of the feeble-minded; they seek to exercise a guiding influence in the problems of education; they essay to deal more efficiently with delinquency; and in the wide realm of human behaviour they set out to help those who, by reason of their inability to make satisfactory adjustment to their environment, are rendered unhappy and socially inefficient." To say that mental hygiene is important to the community is to say something which is quite obvious. However, in a recent report issued by the World Health Organization, we are reminded that in 1950 its Expert Committee on Mental Health stated that the most important single long-term principle for the future work of the World Health Organization in the fostering of mental health was the encouragement of the incorporation into public health work of the responsibility for promoting the mental as well as the physical health of the community.

The promotion of mental hygiene as part of a country's public health activities is something which possibly has not dawned on many people. This point of view and some of its implications may be considered in the light of a report, one of the World Health Organization's "Technical Report Series", on the Second Session of the Expert Committee on Mental Health held at Geneva in September, 1950. This document bears the date April, 1951. At the outset the committee states its conception of mental health. To define mental health negatively as a state in which the individual is free from certain disorders or characteristics is unsatisfactory; on the other hand, to maintain that adaptation to environment is in all circumstances a mentally healthy phenomenon is to ignore the fact that an environment may be such that the healthy response is an attempt to change it. Mental health is stated to be influenced by both biological and social factors. It is not a static condition, but subject to variations and fluctuations of degree; the committee's conception implies the capacity in an individual to form harmonious relations with others, and to participate in, or contribute constructively to, changes in his social and physical environment. It implies also his ability to achieve a harmonious and balanced satisfaction of his own "potentially conflicting instinctive drives". It further implies the development of an individual's personality in a way which enables these drives to find harmonious expression in the full realization of what he is capable of. So much for the definition of mental health. The committee uses the term mental hygiene in its strict and literal sense, analogous to the way in which the general term hygiene is used in public health practice. The practice of mental hygiene demands that individuals should examine and assess their interpersonal relationships in the light of their influence on the development of personality and on mental health. In order that this may be accomplished, education in theory alone is insufficient. Education must be accompanied "by practical methods of learning through actual personal and group experiences which foster emotional insight and modify behaviour in the direction of healthier personal relationships and healthier personality development". Sections of the report are devoted to maternity services, the infant and the pre-school child, separation of the pre-school child from the mother,[1] school health, the handicapped child, communicable diseases and the care of the aged. We do not propose to refer to any of these questions in detail, but rather to draw attention to what is written about the health education of the public and the training of those who are to undertake the education.

In this matter of mental hygiene and the public health services, such subjects as maternity services, the infant and pre-school child, the handicapped child and so on cannot be ignored. It is in the early years that the foundations are laid of mental health in later life. A mental hygiene educator in the field of public health will therefore have as his first care the welfare of mothers in their pregnancies and of children in their infancy. For this he has to be specially equipped. He will not deal with the condition of infants and children in the same way as he will deal with those of more mature years whose mental health is lacking. A great deal of this report is an academic statement on public health administration and on the mental hygiene training to be given to public health workers. One important point is made. Mental hygiene is mentioned as part of the basic training of all types of public health personnel, and then reference is made to the

[1] This subject was discussed in the light of another publication from the World Health Organization in the issue of August 25, 1951.

training of public health specialists in mental hygiene. Before public health specialists can be trained in a country, it is necessary for that country to develop public health general psychiatry and the application of mental hygiene principles. The committee thinks that the training of existing public health workers in mental hygiene is of immediate practical importance. We know that as one star differs from another star in glory, so one public health department may differ from another, and individual members of that department may differ from each other. To emphasize the importance of mental hygiene to officers of health departments is not to imply that the officers of all health departments are devoid of knowledge or experience in mental hygiene. As a matter of fact, public health medical officers are or should be the apostles of preventive medicine, and as such they must concern themselves with the whole of man, with his mind and his body. We may thus agree that if set courses in mental hygiene are to be provided for the average public health worker, certain essentials should be observed. Emphasis should be placed on the understanding of normal rather than of abnormal reactions. An effort should be made to understand the emotional forces concerned in interpersonal relationships, and the relation of anxiety to emotional energy. The mechanisms for the handling of anxiety must be understood, as well as the relationship between the patient and the health worker.

At the end of this discussion the committee refers to what it calls socio-dynamic factors. These are "the external factors in the life situation which have a bearing on mental health". Three headings are included here—family, social forces and cultural background. These are probably more important than anything that can be mentioned in connexion with mental health. In regard to the family, we know that when family life is satisfactory mental health is likely to be good; but today family life, like many human institutions, has deteriorated. This is no doubt the result of what are described in the report as social forces. They include economic, occupational, religious, educational and recreational considerations. The cultural background is, of course, closely bound up with these. The education and religious training of a child are the central pillars on which mental health must be built. If the foundations of these pillars are solidly laid, there should not be much difficulty in regard to mental health. It is when they have been neglected that difficulty arises. A person who is amoral or who has been brought up in an atmosphere in which crime and other forms of delinquency such as prostitution are rife, cannot be expected to display what may be called rude mental health. A person in this position cannot be expected to lift himself out of his unfortunate surroundings—he probably will not regard them as unfortunate. He is like the "man with the muckrake" of John Bunyan—he cannot lift himself out of the mire by the nape of his own neck, as it were. Before any progress can be made, or rather, before any start can be made in the regeneration of such a person, some philosophical basis of education and religion must be established. This will seldom be the task of the public health worker as such. To go further into this vast subject in the present instance does not appear desirable. The conclusion of the present discussion must be that public health workers are only one of those groups of persons who should make the teaching and practice of mental hygiene part of their duties. The World Health Organization, by its Expert Committee on Mental Health, has shown public health workers the enormous extent of the ground to be covered and some of the ways in which an attempt may be made to cover it. The report clearly will be useful to all those who are concerned in this subject.

Current Comment.

THE LIFE OF RED CELLS IN LEUCHÆMIA.

It would be interesting to know how much fundamental information on normal physiological processes has proceeded from study of the abnormal. Perhaps one of the reasons why leuchæmia has attracted so much attention is that its understanding, incomplete as this is, is wrapped up in such comprehension as we possess of the life and career of the formed elements of the blood. Researchers who start out to obtain specific evidence about certain hypotheses are not always bent on a fruitful inquiry, but Ragnar Berlin, in looking for experimental evidence in support of beliefs concerning the life span of red cells in leuchæmia, has obtained interesting results, and has encouraged similar studies.[1] Some of the known facts about leuchæmia suggest that a latent hæmolytic state or syndrome may lie at the back of the occurrence of a normocytic anæmia of varying degree in this disease. These are the urobilinuria, the reticulocytosis and the erythropoiesis which accompany the affection of the red cells in the circulating blood. It used once to be taught in a loose sort of fashion that the vast crowding of the bone marrow with precursor white cells embarrassed the development of the young red cells, but the presumptive evidence leads us to look more deeply.

Eric Ponder, in a succinct analytical review of hæmolytic anæmia in special relation to the hæmolytic mechanisms involved,[2] has pointed out that the red cell may disappear after fulfilment of its destiny in several ways: by fragmentation, by phagocytosis, or by hæmolysis. Of course, it does not follow that manifestations of these processes would be evident in both normal and abnormal conditions. Both these writers accept Ashby's technique as reliable and versatile in research of this kind. This method has cleared up the question of the life span of the red corpuscle by transfusing blood of one group—that is, group O—into a recipient of another group, the cells of both groups being serologically compatible and recognizable. After agglutination of the recipient's cells by an anti-A serum the surviving cells of the donor may be counted, and thus by serial examinations the survival time of the transfused cells may be determined. This assumes that the transfused cells are as well treated by the recipient as his own, an act of biological gratitude which is now a basic element of daily hæmatological practice. Berlin used the original Ashby technique, but employed blood from a finger tip instead of from a vein. He tested the method for accuracy and found it consistent and reliable, provided the anti-serum was really potent. The clinical material he used included both the normal and the abnormal. He agrees that the normal life span of the red cell is about 120 days. His controls numbered 25, and as he had observed that the elimination curve was steeper in the premenstrual period in women, he also examined two women who had had oophorectomy performed. He was not able to assemble sufficient material to arrive at any explanation of this phenomenon. This work was prefatory to an examination of 15 patients with myeloid, and nine with lymphatic leuchæmia; this part of the

[1] *Acta medica Scandinavica*, Supplement 252, accompanying Volume CXXXIX.

[2] *Blood*, June, 1951.

research the author describes in detail. In these patients the most interesting findings were that in the myeloid cases a moderate reticulocytosis was always found when the spleen was markedly enlarged, the mean corpuscular volume was also increased, and a similar association with splenomegaly was found in the excretion of urobilin in the urine. When the spleen was not notably enlarged the red cells showed no abnormality in their life span, but a shortened life and a large spleen were associated. Further, the author found that a curtailed life of the red cells was of paramount importance in the development of anæmia, and that red cell counts would remain at a satisfactory level, maintained by the erythropoietic activity of the marrow, until increasing destruction outpaced the production of new cells. In lymphatic leuchæmia, on the other hand, the encroachment of the lymphatic cells on the marrow seemed to be of importance. There is hardly room here to follow Berlin in his discussion of the exercise of the functions of the spleen under conditions such as prevail in leuchæmia associated with great splenomegaly, nor can we linger over the literature dealing with the results of splenectomy in myeloid leuchæmia, though the record series of the Mayo Clinic still remains fresh in the mind, even though it was published twenty-three years ago. In Berlin's own series, six patients with the myeloid type of the disease were submitted to splenectomy and one with lymphatic. The end results so far observed are equivocal. The red cell survival rate was increased in several patients, and two patients are still alive, one very well and the other fairly well. The occasional long history of myeloid leuchæmia must be always remembered. The author thinks that remission of the disabling anæmia might be a warrant for this procedure. This brings us back to where we started—the life span of the red cell and the factors influencing it. Ponder, in his review, reminds us that the aging of red cells is not entirely mechanical but is also due to the curtailing of metabolic processes, in particular those related to enzymatic activities. He further remarks that we must take into consideration processes seen under abnormal conditions as well as those that are routines of physiological life. Thus, Ponder stresses the non-lytic nature of plasma in itself, but points out that there are lysin-inhibitor-accelerator complexes which may be freed by various influences from their loose binding and may confer lytic activity upon the plasma. So a hæmolytic episode may occur when the balance of the components of a complex is lost and the rate of destruction of red cells rises above that of their production. The form of the Ashby curve may give information about the nature of the precipitant of hæmolysis, whether intrinsic or extrinsic. A drug may initiate such a process, or an agglutinin which may be of immune origin and may or may not require complement to activate it. In passing, some of the Australian work on the hæmolytic action of certain snake venoms may be recalled. Finally, intrinsic defects of structure may be involved; we may not see sickle cell anæmia in Australia, but the Mediterranean types of anæmia may yet appear with a mixture of races. These conditions have no connexion with leuchæmia, but all are linked by at least one aspect of life—the viability of the erythrocyte.

HEMISPHERECTOMY IN THE TREATMENT OF INFANTILE HEMIPLEGIA.

Last year Rowland A. Krynauw, of Johannesburg,[1] reported his results in 12 cases of infantile hemiplegia treated by means of cerebral hemispherectomy, an operation that he has been performing since 1945. Krynauw states that local excisions of cortex may stop convulsions in cases of infantile hemiplegia, but they have no beneficial effect on the mental state of the patient, nor do they lessen physical disability. Because of this fact he was led to the more radical procedure of removal of the affected hemisphere with the exception of the thalamus, the caudate nucleus and its tail. Convulsions and mental changes were regarded as indications for operation. The ages of the patients concerned varied from two to twenty-one years. Krynauw reports that after operation epilepsy, either focal or generalized, ceased without sedative medication. The patients improved greatly in relation to any disorder of behaviour or personality previously present, an improvement that Krynauw attributes to the normal functioning of the remaining hemisphere once it has been freed from the influences of the abnormal side. Return of motor function and power to pre-operative level and in some cases improved tone and function beyond this level have been obtained. If, however, the caudate nucleus is removed with the hemisphere, there is little or no return of useful motor activity. On the sensory side Kraynauw has found that localization of pin-prick returns after a few months with only a minimal defect of accuracy. Two-point discrimination with an increased threshold (about three times the normal) has been present in two cases. Joint sense is impaired. There is an entire absence of those changes, such as spatial disorientation and disturbance of body image, that are usually associated with disturbances of the parietal cortex.

It was pointed out recently by Sir Hugh Cairns[1] that previously to Krynauw's work cerebral hemispherectomy had been performed for infiltrating glioma by Dandy (who reported this in 1928) and by others. Moreover, the operation was actually carried out in a case of infantile hemiplegia by Kenneth McKenzie in Toronto in 1938. However, it is to Krynauw, Cairns asserts, that "credit should be given for realising the value of this operation and developing it into a practical treatment for infantile hemiplegia". Cairns himself reports his experience with the operation in three cases. He describes his results, assessed a year after hemispherectomy, as dramatic. The patients' convulsions have stopped and the hemiplegia is no worse—indeed, he states, in two cases the patients' condition is a little better in this regard because the muscles are less spastic. By far the most remarkable improvement is in mentality. The patients' behaviour was examined before and after operation by a psychologist, Miss M. A. Davidson, who records her findings in detail in a supplement to Cairns's paper; they are generally favourable and are summarized conservatively in the conclusion that the patients "are happier and more adequate people as a result of their operations a year ago". Cairns sums up by stating that his experience confirms Krynauw's claim that hemispherectomy works a remarkable transformation in these patients; it not only stops their convulsions, but, still more important, greatly improves their mental state. Temper tantrums cease, and the patients become happy and good-tempered and begin at once to show intellectual and sometimes artistic development. They can go to school, or if adults they become employable, but their speed of intellectual performance, though better than before operation, is still slower than normal. Naturally, Cairns points out, the result obtained is influenced by the patient's pre-operative personality.

What ultimate place this extremely radical procedure is to take in the treatment of infantile hemiplegia it is yet too soon to predict. Cairns considers that more experience is needed before the indications for cerebral hemispherectomy can be fully defined. It is not suitable, he states, for all cases of infantile hemiplegia, but it seems clear that it should be undertaken in any case of established infantile hemiplegia with fits which cannot be controlled by drugs, or in which there is backwardness or deterioration in the patient's intellectual or emotional state. Before operation it is essential to establish by careful clinical examination and air studies that one cerebral hemisphere is diseased and the other healthy. As with other major procedures in psychosurgery, many people who are entirely favourably disposed to progress will still feel constrained to watch the trial of cerebral hemispherectomy with a conservative eye. If it proves itself effective as a measure in the management of infantile hemiplegia, and if it does not raise problems larger than those it solves, it must become established.

[1] *Journal of Neurology, Neurosurgery and Psychiatry*, November, 1950.

[1] *The Lancet*, September 8, 1951.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

The Adrenal Cortex and Treatment with Pyrogenic Substances.

WILLIAM ARENDSHORST AND HAROLD F. FALLS (*Archives of Ophthalmology*, November, 1950) have examined the response of the eosinophile cell count to the administration of typhoid H antigen in the treatment of ocular disease, in order to see whether the effects of protein therapy depend on the release of 11, 17-oxysteroids from the adrenal cortex. They state that in the cases studied dosage of antigen administered was 15,000,000 to 25,000,000 units initially, and this dose was increased by 10,000,000 to 20,000,000 units every other day depending on the response of the patient. There was a correlation of eosinopenia, fever and general response when typhoid H antigen was given intravenously; the eosinopenia and adrenocortical function are generally related directly to the degree of fever and malaise. Failure of improvement after an initially good adrenocortical reaction is explained by inhibition of the hypothalamic-pituitary axis by the abnormally high titre of 11, 17-oxysteroids. As compared with ACTH or cortisone therapy foreign protein therapy has advantages and disadvantages. The most obvious advantage is its low cost, and its great disadvantage is rapid development of tolerance to its effects, a tolerance which is not overcome by changing the pyrogen. Foreign protein has been found effective as a supplementary therapeutic agent after withdrawal of a patient from ACTH therapy. When patients become refractory before their disease is controlled, when a prolonged course of treatment will be necessary, and when patients fail to show an eosinopenic response to adequate doses, the use of ACTH or cortisone should replace foreign protein therapy.

Surgery of the Lens in Infancy and Childhood.

PAUL A. CHANDLER (*Archives of Ophthalmology*, February, 1951) discusses the surgery of cataracts in infancy and childhood. He enumerates the indications for surgery, the various procedures available, the expected complications, and how these complications may be avoided. He is of the opinion that if the pupil cannot be adequately dilated before operation then linear extraction with complete iridectomy should be performed. If pupillary dilatation is good before operation, then a peripheral iridectomy will suffice. For cataract associated with uveitis the author recommends intracapsular extraction.

Topical Cortisone Treatment for Anterior Segment Eye Disease.

E. H. STEFFENSEN *et alii* (*American Journal of Ophthalmology*, March, 1951) report their findings in the treatment of anterior segment eye disease with topical cortisone therapy. The patients were treated in the out-patient department. The cortisone solution was made up by diluting one volume of the saline suspension of cortisone with four volumes of normal saline. The solution was instilled into the eye every thirty to sixty minutes during the waking period. The authors report 24 cases in detail, including non-specific superficial and deep keratitis, phlyctenular conjunctivitis, dendritic keratitis, vernal conjunctivitis, and acute iritis with secondary glaucoma. The therapy was found to be ineffective against vernal conjunctivitis, dendritic keratitis, trophic ulcer and Fuch's epithelial dystrophy.

Cycloelectrolysis for Glaucoma.

CONRAD BERENS *et alii* (*American Journal of Ophthalmology*, January, 1951) describe cycloelectrolysis, a new operative technique devised for the purpose of reducing intraocular pressure in various types of glaucoma; it differs from perforating cyclodiathermy primarily in the type of current employed to produce atrophy of part of the ciliary body. In cycloelectrolysis there is a controlled chemical decomposition of tissue which is produced by means of galvanic current. In cycloelectrolysis, the negative pole of a galvanic current attached to a fine needle is used as the "active" electrode, and the positive pole or "dispersive" electrode is placed beneath the patient's shoulder. When the current is applied and the needle penetrates the ciliary body, the sodium chloride, present in solution in all body tissues, is ionized. The sodium ion migrates to the cathode, is neutralized, and then reacts with water to form sodium hydroxide with release of hydrogen. Sodium hydroxide is a strong caustic which causes a chemical liquefaction of cells around the needle. By regulation of the amperage and the duration of the application, the amount of tissue dissolution may be controlled accurately. Cycloelectrolysis is designed to reduce the production of aqueous by chemical destruction and subsequent atrophy of part of the ciliary body and its processes with the least possible trauma and a minimum of complications. Indications for cycloelectrolysis are chronic primary glaucoma, glaucoma in aphakia, narrow angle glaucoma, absolute glaucoma, and uveitis with secondary glaucoma; it may prove beneficial for congenital glaucoma, central vein thrombosis, rubeosis of the iris, hæmorrhagic glaucoma, acute glaucoma and secondary glaucoma from trauma. The procedure requires no special skill and can be performed in out-patient practice.

Lamellar Keratoplasty.

RAMON CASTROVIEJO (*American Journal of Ophthalmology*, December, 1950) describes his technique for lamellar keratoplasty and compares the operation with that of penetrating keratoplasty and keratectomies. He states that lamellar keratoplasty does not give as brilliant results as penetrating keratoplasty. However, lamellar keratoplasty is far safer and avoids unpleasant complications such as iris incarceration and prolapse injury to the lens, secondary glaucoma and endophthalmitis. It is the procedure of choice for one-eyed persons, for aphakia and for all superficial corneal opacities, particularly those affecting the pupillary area with good surrounding cornea. It is also to be preferred when post-operative cooperation is expected to be poor or convalescence stormy. If keratectomy offers a fair chance for visual improvement, in cases in which a choice is possible, it would seem to be the preferred operation. Lamellar keratoplasty is justified whenever there are no other effective means of treating certain corneal conditions such as Mooren's ulcer, recurrent herpetic rosacea, and neuroparalytic keratitis.

Local and Systemic Cortisone Therapy in Ocular Disease.

IRVING H. LEOPOLD *et alii* (*American Journal of Ophthalmology*, March, 1951) have conducted a clinical study on 142 eyes treated with cortisone administered by subconjunctival injection, retrobulbar injection, and systemic and topical application. As a subconjunctival injection the dosage used was 0·05 millilitre of cortisone suspension given daily for three days, repeated after two days. For topical application the strength employed was 1:4 or 1:8 in normal saline. When used as a retrobulbar injection the dosage was 50 milligrammes in two millilitres with 0·5 millilitre of 4% procaine solution. When systemic therapy was used dosage was 100 milligrammes given every eight hours for three doses, 100 milligrammes every twelve hours for two doses, and then 100 milligrammes daily. The drug failed to produce improvement in ocular pemphigus, *erythema multiforme*, corneal dystrophy, congenital luetic interstitial keratitis, uveitis due to Boeck's sarcoid, Vogh-Koyanagi syndrome and Harada's disease. The authors state that topical cortisone therapy will relieve blepharitis, allergic conjunctivitis, vernal conjunctivitis, episcleritis and some cases of keratitis. Subconjunctival cortisone therapy is beneficial in cases of episcleritis, a variety of corneal inflammations and anterior uveitis. Cortisone when injected in the dosage used by these workers produced no irreparable damage; after subconjunctival injection there is some reaction and the cortisone remains as a yellowish mass which takes five to seven days to disappear. After retrobulbar injection gross œdema may be expected. In a definite number of cases improvement occurred only while cortisone was being administered; another group of patients improved temporarily and subsequently failed to benefit from further therapy.

Operations in Convergent Squint.

PAUL A. CHANDLER (*American Journal of Ophthalmology*, March, 1951) considers the surgical treatment of monocular and alternating convergent squint. He states that for monocular squint, operations are usually performed on the squinting eye. In all cases in which there is a good near point, that is, 30 millimetres or less, the internal rectus should be recessed, whether or not resection of the external rectus is carried out. If the near point is remote the internal rectus should not be recessed; resection of one or both external rectus muscles should be carried out. If there is profound amblyopia with no possibility of binocular vision, one should aim at under-correction. In general, surgery should be more conservative if there is a high degree of hypermetropia. In most cases of alternating squint there is apparent overaction of the medial recti, there may or may not be an accommodative element, fixation is homonymous, and the near point is good. The author recommends recession of both medial recti, recession of one medial rectus, or recession of one medial rectus and resection of the lateral rectus of the other eye. He

considers that recession of the medial rectus and resection of the lateral rectus of the same eye (especially if there is an accommodative element) are unsatisfactory, even though the cosmetic result may be good. Recession of one medial rectus and resection of the lateral rectus of the other eye is satisfactory in squint up to 30 or 40 dioptres. It is not indicated when the squint is small for distance and quite large for near vision. Here, bimedial recession is preferable. In cases of underaction of the lateral recti with little or no secondary contracture of the medial recti there is crossed fixation. The near point is poor. The operation of choice is resection of both lateral recti. A larger resection may be carried out on one eye than on the other if one lateral rectus appears to be weaker than the other. When resection is performed alone the effect is much less than if the medial rectus is recessed at the same time. When there is underaction of the lateral recti with contracture of the medial recti, a condition usually found in early infancy, the medial recti are strong and inelastic and give rise to the inference that both lateral recti are paretic. Recession of both medial recti is the operation of choice, followed by use of glasses with the nasal two-fifths of each lens occluded. In these cases the child should be operated upon early in life. When there is overaction of the medial recti with underaction of the lateral recti, recession and resection in one eye, recession of both medial recti, resection of both lateral recti and recession of the stronger medial rectus combined with resection of the lateral rectus of the other eye, have all been satisfactory.

OTO-RHINO-LARYNGOLOGY.

Speech Audiometry in the Assessment of Deafness.

E. D. D. DICKSON AND D. L. CHADWICK (*The Journal of Laryngology and Otology*, August, 1950) state that free-field testing, in which the patient faces a loud-speaker about four feet from its centre, more nearly represents normal hearing conditions than methods employing headphones. Since wide variations are found to occur in articulation scores when the directly amplified voice of trained speakers is employed, high-fidelity recorded speech which is reduced to a uniform intensity is considered to test the hearing capacity more accurately. A normal speech audiogram was obtained by testing 100 people whose hearing was shown to be normal by pure tone audiometry. With this normal curve as a base line, speech audiograms have been plotted for more than ninety deaf patients. The curves are plotted to show the average percentage articulation score at intensities ranging in five decibel stages from 20 decibels to 65 decibels. In cases of nerve deafness it is shown that above a certain intensity no further increase in intelligibility is obtained by further increasing the amplification. Speech audiometry has proved an extremely satisfactory method of comparing the results obtained when different hearing aids are worn. The average improvement obtained was 9·7 decibels. Thirty-five patients who had undergone fenestration were tested before and after operation. It was found that the pre-fenestration and post-fenestration curves were not always comparable in shape, there being a plateau about the middle of the curve in many after operation and frequently also a high intensity dip. A detailed analysis has been made of the results obtained with 35 fenestrated otosclerotics by comparison of pure tone and speech audiograms. The "average decibel improvement" at 512, 1024 and 2048 cycles is used and is compared with the average gain for speech. This comparison shows more optimistic results given by pure tone audiometry than those given by speech audiometry (20·5 and 9·8 decibels respectively). The results given by speech audiometry appeared to correspond more closely to the patients' own observations of improvement. The speech audiogram may, however, show progressive improvement tending in time to approach the pure tone result, presumably owing to re-education of the faculty for hearing and interpreting of speech.

Factors Influencing Late Secondary Hæmorrhage following Tonsillo-Adenoidectomy.

W. K. WRIGHT AND L. G. PRAY (*Archives of Otolaryngology*, March, 1951) state that possible factors influencing late secondary hæmorrhage after tonsillectomy are acetylsalicylic intake, vitamin *K* and *D* deficiencies, excessive surgical trauma, infection, and blood dyscrasias. Of these, post-operative administration of acetyl-salicylic acid has been suspected of being the commonest cause. Some writers have reported no instances of hæmorrhage when aminopyrine was given post-operatively instead of acetyl-salicylic acid. Others have reported a beneficial effect from administration of vitamin *K* and of vitamin *C* on salicylate-produced hypoprothrombinæmia. The present series consists of 1648 cases of tonsillo-adenoidectomy. The first 411 were used to study the effect of vitamin *C* on secondary bleeding. In the remaining 1237 cases a study was made of the effect of elimination of acetylsalicylic acid. In the first group free use of acetyl-salicylic acid was allowed to all patients, but alternate patients received in addition vitamin *C* in appropriate dosages, the remainder serving as controls. The vitamin *C* did not appear to have any anti-hæmorrhage influence. In the second group in which the patients took no acetylsalicylic acid, but were given supplementary doses of vitamin *K* and vitamin *C*, the secondary bleeding was only one-tenth as much as in patients who had had acetylsalicylic acid. The elimination of this substance is thus considered to be a significant factor in the control of late secondary hæmorrhage. On the basis of the figures studied there was no evidence that administration of vitamin *K* reduced the incidence of secondary hæmorrhage.

The Otological Concept of Bell's Palsy and its Treatment.

J. A. WILLIAMS AND J. B. SMITH (*Annals of Otology, Rhinology and Laryngology*, December, 1950) state that 75% of patients with Bell's palsy recover spontaneously. The 25% who do not recover are forced to go through life with disfigurement. Pressure on a nerve influences its conductive capacity by means of local ischæmia. The arteries which supply a nerve divide into ascending and descending branches which course in the epineurium. Some branches pass deep into the nerve and run between the fasciculi. The vessels are arranged predominantly in a longitudinal pattern. The infrafascicular plexus is continuous throughout the length of the nerve, being fed or reinforced at various levels by the nutrient arteries in such a way that no one nutrient artery may be considered as dominating any portion of this plexus. The intraneural vascular bed of a nerve therefore is a continuous longitudinal system. When compression involves a nerve which lies in direct relationship with a bony surface, such as the Fallopian canal and stylo-mastoid foramen, ischæmia may be complete, so that there is a widespread disturbance of motor activity. During the decompression operation for Bell's palsy the nerve fibres are seen to bulge forth slowly when the pressure is released by slitting the sheath. Paralysis has been speedily relieved after slitting of the sheath down to the stylo-mastoid foramen. The cause of the œdema is unknown. At the stylo-mastoid foramen the bony diameter is much smaller than that of the Fallopian canal, in which the nerve occupies about two-thirds of the space. At the foramen the contents are firmly bound down by a fibrous tissue sheath. Any swelling from within would probably affect the venous return with blocking of the lymph drainage, the relatively narrower foramen acting much as an experimental clip or ligature around the nerve has been shown to act in experiments on animals. A case of Bell's palsy may well be due to dysregulation of the arterial supply of the facial nerve or localized thrombophlebitis. Recovery after decompression of the nerve may be explained on the basis of permitting early and effective collateral circulation. Early decompression of the nerve is advocated for Bell's palsy, especially if accompanied by pain and when absence of a faradic response is revealed. When the faradic response is inconclusive by testing through the skin, it is suggested that direct exposure of the nerve should be carried out to establish with certainty the presence or absence of this reaction.

Radiocobalt in Otolaryngology.

LEWIS F. MORRISON (*Archives of Otolaryngology*, February, 1951) states that radiocobalt is an effective, efficient and inexpensive source of γ radiation, comparing more than favourably with radium; β radiation is weak and easily screened. The metal is cheap and can be fabricated into any shape prior to activation. The production of radiocobalt in the form of beads provides the advantage of closer approach to a "point source" of radiation. The beads are fixed in the end of a Foley catheter and can be readily secured in the desired place by the use of the inflatable cuff. The method has been used in the treatment of 11 patients with malignant neoplasms of the nasal regions. The initial local reactions are similar to those obtained after exposure to similar doses of radium. The period of observation is much too short to permit evaluation of results. Since the γ radiation from radiocobalt is similar to that from radium, similar results are to be anticipated.

British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney, Crow's Nest, New South Wales, on June 21, 1951. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staff of the hospital. Parts of this report appeared in the issues of September 22 and 29 and October 6 and 13, 1951.

Left-Sided Colonic Carcinoma of the "Adenoma Destruens" Type.

Dr. T. F. Rose discussed the case of a man, aged sixty-nine years, who had had a history of four months' anorexia, diarrhœa, vomiting and loss of weight. A large movable mass had been found in the left hypochondrium disappearing up behind the costal margin. The lower pole was smooth, firm and rounded. In hospital the patient had an intermittent fever up to 101° F. with a leucocytosis of 13500 per cubic millimetre, 90% being neutrophile cells. A barium enema flowed up only to the left iliac crest where it became obstructed. A little barium got beyond the point showing destruction of the mucosal pattern of the descending colon. The mucosal pattern was broken up and bizarre. Laparotomy revealed a very large carcinoma of the colon, involving the descending colon from the splenic flexure to the iliac crest. It was a fungating tumour, growing into the bowel lumen, of the type called *"adenoma destruens"*, more commonly found in the right side of the colon. The tumour was invading the bowel wall, and at one point it was attached to the greater curvature of the stomach. Microscopic examination confirmed that it was an adenocarcinoma. An adjacent lymph node was not involved. The mass was resected, and the bowel ends were exteriorized. The patient died some twelve hours later in spite of pre-operative and post-operative antishock measures. At autopsy no secondary deposits were discovered. A pancreatic cyst adenoma was an accidental finding.

Cystic Thecoma of the Ovary.

Dr. Rose next presented a girl, aged fourteen years, who fifteen months before her admission to hospital had had her first menstrual period, in which she lost a little blood only. Ever since then she had had a thin yellow discharge, but no further menstrual periods. Fourteen months later she had noticed that her abdomen was swelling. It enlarged rapidly, and in the ten days prior to admission to hospital she became very breathless and the swelling became increasingly painful. Examination of the patient disclosed a thin pale girl with an enormously swollen abdomen. She was dyspnœic and had to sit up in order to breathe. The abdomen was filled with a tensely cystic mass stretching from the pubis to the xiphisternum and into both flanks. Rectal examination gave little information except that the cervix was in its normal position. The patient was developed normally for her age, and there was no evidence of sexual precocity. The breasts were normal in size, and the distribution and extent of the pubic and axillary hair were normal. The external genitalia were normal. An Aschheim-Zondek test yielded a negative result. At operation, a few hours after the patient's admission to hospital, a thick-walled right-sided ovarian cyst containing 120 fluid ounces of clear, somewhat brownish fluid was removed. It was unilocular, and its walls were smooth. There were no thickened areas in the walls. The uterus and Fallopian tubes and the left ovary were normal. Convalescence was uneventful. Dr. Rose said that the histological picture showed the cyst wall to have the structure of a thecoma, which was a rare, usually solid ovarian tumour occurring at a later age period. Novak suggested that the granulosa cell and thecal cell tumours of the ovary should be classified under the terms feminizing mesenchymoma of the ovary, as all those cells were from the ovarian mesenchyme. The tumours might be associated with endocrine disturbances, such as precocious puberty, if they occurred before puberty; before the menopause they might cause menorrhagia or even amenorrhœa, and after the menopause they might cause post-menopausal bleeding.

Small Bowel Obstruction following Resection of the Rectum.

Dr. Rose's next patient, a man, aged eighty-four years, had ten months previously undergone abdomino-perineal resection of the rectum for adenocarcinoma. All had gone well save for a stricture of the colostomy opening at the junction of skin and mucosa. Though the opening would hardly admit a finger, yet the condition caused no inconvenience to the patient until twenty-four hours before his admission to hospital when he suffered severe abdominal colic with complete constipation and vomiting. On examination of the patient, there was a little lower abdominal distension with some tenderness. The colostomy admitted the finger tip with difficulty. A few borborygmi were present, but not appreciably accentuated. X-ray examination of the abdomen revealed dilated small bowel with numerous fluid levels. Laparotomy showed that the ileum was obstructed in two places; one obstruction was due to an encirling band and one to an adhesion fixing the loop to the reconstituted pelvic floor. The second obstruction was two feet from the caecum. The bands were divided, and in addition a round mass two inches in radius was found in the mesentery of the affected loop. It was removed and seen to be not a secondary carcinoma but a lipoma. Convalescence was uneventful.

Infection after the Plating of a Fracture of the Femoral Shaft.

Another patient presented by Dr. Rose was a man, aged twenty years, who four years previously had been involved in a motor-cycle accident in another State. That resulted in a simple transverse fracture of the shaft of the right femur, which was plated, but infection supervened so that the plate had to be removed. However, a large piece of the cortex to which the plate was secured eventually sequestrated, although the fracture itself united. The patient was examined at the Royal North Shore Hospital of Sydney four years after the accident, when he had numerous discharging sinuses leading to the fracture site. There was only a 90° range of involvement of the knee joint from full extension due to quadriceps fibrosis. X-ray examination disclosed the large sequestrum, which was removed. (Dr. Rose exhibited it at the meeting.) The wound was sutured and healed by first intention with penicillin therapy.

Traumatic Extraperitoneal Rupture of the Bladder.

A little girl, aged five years, shown by Dr. Rose, had been knocked over by a motor-car so that one wheel ran across her abdomen. On admission to hospital one hour later she was very shocked. There was generalized abdominal tenderness with rigidity greatest in the suprapubic region. On urethral catheterization pure blood was withdrawn from the bladder. X-ray examination disclosed fractures of the bodies of the pubes on either side of the symphysis in good position. There were no fractures posteriorly. The shock was treated by blood transfusion, and two hours after admission the patient was fit for operation. That disclosed much blood clot and urine in the cave of Retzius and a large tear in the infero-lateral surfaces of the bladder just above the neck. The back of the pubis was roughened where the fractures were. Exploration of the peritoneal cavity through a small opening in the peritoneum above the bladder showed no evidence of intraabdominal injury. A small Malecot catheter was placed in the bladder through the tear which was too deeply situated to suture. Convalescence was uneventful save for the occasional passage of blood clots *per urethram*. The catheter was removed on the tenth day. Normal micturition quickly occurred, and the sinus closed. When examined nine months later the patient had had no further trouble and was very well. Dr. Rose said that from the situation of the lesion in the bladder, it was probable that the pubes had been forced inwards, so tearing the bladder, They had then sprung out when the weight was removed.

Severe Head Injury.

Dr. Rose also showed a man, aged twenty-five years, who had been admitted to the Royal North Shore Hospital of Sydney on May 7, 1951, after having been involved in a motor-cycle accident. While attempting to turn he had caught the wheel of the cycle in the tram lines and been thrown, striking his head violently against a telegraph post. On admission to hospital he was unconscious and in a state of cerebral irritation. Examination revealed bruising and hæmorrhage in both orbits; blood was present in the mouth, nose and left ear. The left pupil was larger than the right, and did not react to light. Conjugate movements of the eyes were observed when the eyelids were opened. A Babinski plantar response was present on the right side. The blood pressure was 130 millimetres of mercury, systolic, and 80 millimetres, diastolic; the pulse rate was 56 per minute. Shortly after admission he vomited about 15 ounces of dark altered blood. Half-hourly pulse and blood pressure readings were ordered. As he was unable to pass urine an indwelling

catheter was inserted, and he was fed through a Ryle's tube. He regained consciousness five days later, but was still confused and restless, and a subconjunctival hæmorrhage was noted in the right eye. Since then he had improved slowly, although he was unable to pass urine until June 1, 1951. X-ray examination of the skull revealed multiple fractures, the following report being made:

> An extensive comminuted fracture of the skull, the principal limits of which lie on the left side. The fracture involves frontal, parietal and occipital bones on the left side, and involves the left half of the middle cranial fossa. There is also a linear crack in the right parietal bone extending posteriorly from about the mid point of the fronto-parietal suture, and for about one inch vertically into the maxilla. There are fractures of the mandible on both sides. On the right side the fracture lies in the horizontal portion about 1" to the right of the symphysis. On the left side the fracture appears to lie at the root of the condyloid process.

On May 27, 1951, the patient got up and locked himself in a room, remaining there for some time before the nursing staff were able to restore him to his bed. Some hours later his temperature rose to 105·6° F. and his pulse rate to 140 per minute. Lumbar puncture was performed; the cerebrospinal fluid was clear and at a pressure of 190 millimetres of fluid. It contained nine lymphocytes, one polymorphonuclear cell and ten red blood cells per high-power field. The biochemical findings were within normal limits. Two days later his temperature and pulse had returned to normal. Since regaining consciousness the patient had been very confused—for instance, if shown an article, he knew what it was used for but not its name. Five weeks after the accident, retinoscopic examination showed left optic atrophy and a fixed dilated pupil, probably due to severe injury to the optic nerve.

Atypical Sudeck's Post-Traumatic Osteoporosis: Pancreatic Pseudocyst Complicating Pancreatitis.

The other two cases discussed by Dr. Rose were one of atypical Sudeck's post-traumatic osteoporosis of the left leg following fractures of the distal phalanges of the third and fourth toes, and one of pancreatic pseudocyst complicating pancreatitis. These will be fully reported later.

Care of the Premature Baby.

DR. CLAIR ISBISTER and DR. PAULINE GASTON presented three infants, all of whom had been born prematurely and had progressed satisfactorily when given suitable care. In a general discussion of the care of the premature baby it was first of all pointed out, by way of definition, that for statistical purposes every baby of five and a half pounds and less was considered premature and viable if it had two of the following features: a gestation period of twenty-eight weeks, a weight of two and a quarter pounds or more, and a length of 14 inches. Attention was drawn to the fact that in New South Wales, if a baby of under two and three-quarter pounds in weight died, the birth was officially classed as a miscarriage. It needed to be recognized that prematurity was the greatest single cause of neo-natal deaths; therefore prevention of prematurity and care of the premature baby during and after labour offered the best chance of a reduction in the death rate. The care of the premature baby was discussed under three headings: the maintenance of body temperature, the prevention of infection, and feeding and general nursing care.

On the subject of maintenance of body temperature, it was pointed out that a special room was essential. It should be warmed or air-conditioned to a temperature of 75° F., and the humidity should be fixed at 65% or water should be heated in the room. There should be no more than six babies in a ward. The use was discussed of the following methods of heating: (*a*) the closed incubator, for example, the "Humidicrib"; (*b*) the electrically heated blanket with its thermostatically controlled temperature in a lined cot; (*c*) hot-water bottles in a lined cot with a thermometer, care being taken that the bottles could not burn or leak onto the baby. It was pointed out that the cot temperature should be 95° to 100° F. for at least a week if the baby was under four pounds in weight, and after that not below 90° F. Babies under three and a half pounds in weight might need a temperature of 100° to 105° F. at first. A dress of cotton wool or soft flannel was necessary for the baby until the second week if its weight was less than four pounds. Handling and exposure should be reduced to a minimum. The baby should be bathed as little as possible, and sterile lanoline should be used if it weighed less than four and a half pounds.

It was then pointed out that premature babies had very poor resistance to infection, so the utmost care had to be taken to prevent infection. They listed the following six precautions. A strict routine was necessary for the washing of hands. The wearing of masks was essential, and people with colds and skin infections should be excluded if possible. Adequate cot spacing should be observed. Only medical officers and nurses should be allowed in the ward. All infectious and potentially infectious babies should be isolated. A watch should be kept for thrush, which might be fatal, and for other infections.

On the subject of feeding and general nursing care, the following points were made. Handling should be minimal and gentle. Rectal temperatures should be taken every four hours unless the cot temperature was fixed. Bathing and oiling should be minimal; it should be carried out in the cot until the baby was four and a half pounds in weight or two weeks old, and then not more than every third day. Napkins should be changed before or between feeds to prevent regurgitation and inhalation of feed. The eyes should be swabbed daily. The mouth should be inspected for thrush, but not cleaned. The nose should not be cleaned unless it was obviously obstructed. The umbilical cord should be kept dry and left alone. Oxygen or "Carbogen" should be given for twenty-four hours and then as necessary. Stimulants should be given as necessary. Feeding was discussed, first of all in relation to the amount and kind of food. It was recommended that for the first twelve hours nothing should be given by mouth in all cases. After twelve hours, feeding should be commenced with a solution of 5% glucose in one-quarter strength saline; one-half to two drachms should be given three-hourly if necessary for twelve to twenty-four hours. From the age of thirty-six to sixty hours one-quarter strength expressed breast milk should be given in an amount of one ounce per pound alternately with the glucose-saline solution. Full strength expressed breast milk should be given by about the seventh day in an amount of two ounces per pound weight, being increased by the twenty-first day to three or three and a half ounces per pound weight. It was pointed out that neither the fluid nor the caloric requirements for bigger babies were as high as they were for smaller babies. If no breast milk was available, the best alternative was one of the following: whey, which might be given for thirty-six to sixty hours, skimmed lactic acid milk diluted with whey and boiled water to the fluid and caloric requirements, unsweetened evaporated milk, dried milk, modified cow's milk. Three methods of feeding were suggested. Sucking from a bottle was suitable for bigger babies. Tube feeding was necessary for babies under three and a half pounds in weight and for any others who regurgitated or had cyanotic attacks. The technique of tube feeding was very important: the baby should be propped up and on its right side and should not be left for twenty minutes after the completion of the feed; that was to prevent regurgitation and death from asphyxia due to inhalation of the food or later from pneumonia due to the same cause. Pipette feeding was necessary for the first forty-eight hours only. The following additions to the feed were suggested: (*a*) vitamin *D* in concentrated preparation starting at the end of the first week and being worked up to an amount of at least 2000 units per day by the fourth week; (*b*) vitamin *C* starting on the third day with an amount of five to ten milligrammes of ascorbic acid increasing by ten milligrammes daily until fifty milligrammes per day were being given; (*c*) vitamin *K* in an amount of five milligrammes; (*d*) vitamin *B* complex in an amount of one-fifth of a tablet of "Benerva Compound" (Roche) on the third day; (*e*) calcium and phosphorus, starting with syrup of calcium lactophosphate on the fourteenth day in an amount of seven minims per pound daily; (*f*) iron, starting in the sixth week with ferrous sulphate in an amount of one and a half grains working up to four and a half grains daily; (*g*) casein hydrolysate, which might be added, but was not used at the Royal North Shore Hospital of Sydney. Referring to after care, Dr. Isbister and Dr. Gaston said that the baby should be kept in hospital until it weighed five and a half pounds. It should be breast-fed if possible when it was discharged from hospital, and that should be continued for at least nine months. Special attention was required in the first year, as premature babies were more prone to anæmia, infections and congenital abnormalities.

Duodenal Obstruction due to Congenital Abnormalities.

Dr. Isbister and Dr. Gaston then presented three cases of duodenal obstruction occurring over a twelve months' period in the obstetric department. The clinical picture and the abnormality in each case were different. The first two patients had died, and only the history and post-mortem

findings were presented. The third child had progressed very satisfactorily since operation. In the first case the baby had been admitted from a private hospital to the premature ward at the age of three days. It was one month premature and birth weight had been five pounds; it had a history of vomiting since birth. No further history was available. On admission to hospital the child was extremely dehydrated and vomited a large amount of green material soon afterwards. Its condition appeared to be very poor, and it was not allowed into the premature ward but was nursed in isolation. The stomach was washed out, and the child retained a 5% solution of glucose in half-strength physiological saline when given both by mouth and rectally. The dehydration became much less during the next twenty-four hours. There was no vomiting and no passing of meconium, but the child's condition appeared much better. During the night it vomited a large amount of green material, some of which it appeared to inhale; then it became very cyanosed and died. From post-mortem examination the cause of death appeared to be inhaled vomitus. No abnormality was present in any organs except the intestinal tract, where the stomach and duodenum were dilated and the rest was collapsed. There was no band or malrotation, but when a probe was passed the duodenum was found to be obstructed completely by a diaphragm of mucous membrane.

In the second case the baby was a full-term female weighing seven pounds one ounce at birth; the delivery was normal. The mother was an elderly *primipara* whose pregnancy had been normal except for mild pre-eclamptic toxæmia. The baby had gone to the breast normally until the fifth day when it vomited yellowish material not unlike colostrum. Small amounts were vomited again on the sixth and seventh days, but the stools were normal and vomiting appeared to subside after twenty-four hours of cot feeding. The baby was discharged from hospital on the eighth day to a convalescent home. She then weighed six pounds seven ounces and was being partially breast fed. She was readmitted to the Royal North Shore Hospital of Sydney two days later still vomiting; the vomitus was said to resemble egg yolk. She was 13 ounces under her birth weight and had not passed a stool for two days. Her condition appeared satisfactory on admission to hospital. Feeding was carefully observed, and occasional peristaltic waves and projectile vomiting were seen. Clinically the diagnosis appeared to be duodenal obstruction, partial or intermittent, and that was confirmed radiologically by barium meal examination. Whey and 5% glucose solution were retained, but breast milk or skimmed lactic acid milk was vomited. Laparotomy revealed, firstly, obstruction of the second part of the duodenum by kinking, and secondly, a freely mobile gut with a "bunch of grapes" appearance and malrotation. The bowel was rotated through 360°, and the obstruction was relieved. The baby appeared to be much better and stopped vomiting for two days; then the symptoms recurred. A second laparotomy was carried out four days after the first, and gastroenterostomy was performed. The symptoms continued, a fistula developed and the baby was kept alive by intravenous feeding for twelve days before it died. Post-mortem examination confirmed the operation findings.

In the third case the patient was a male child, the second child in the family. He had had a birth weight of seven pounds seven ounces. A normal pregnancy had terminated in a normal vertex birth. The child had appeared normal at birth and continued so for the first three days. On the fourth day he became jaundiced, but passed meconium normally and went to the breast normally. On the fifth day he vomited a large quantity of green material. The stomach was washed out and the baby was cot fed for twenty-four hours, at first with breast milk, which was vomited, and then with 5% glucose solution, which was retained. On the seventh day the baby developed pustules and balanitis and appeared very ill. Penicillin therapy was commenced. In the afternoon the baby again vomited green material. On the eighth day barium meal examination revealed a partial obstruction of the duodenum. Observations of the child during feeding revealed no peristaltic waves, and the vomiting was not projectile in type. Dr. L. S. Loewenthal on consultation recommended and carried out immediate laparotomy. This revealed a short duodenocolic ligament causing obstruction and malrotation of the bowel. The constricting band was divided and the gut rotated into a normal position. Blood transfusion was started during the operation, 120 millilitres being given, followed by 5% dextrose solution. Fluid requirements were calculated as three ounces per pound of body weight for the first twenty-four hours, to compensate for dehydration and blood loss, then one and a half ounces per pound for the next two days, slowly increasing thereafter. Intravenous administration of fluids was stopped after sixteen hours. Streptomycin, 70 milligrammes twice a day, and penicillin, 75,000 units of the procaine preparation daily, were given for five days. The baby progressed very well, but for two weeks was unable to tolerate feeds larger than one and a half ounces without vomiting, so he was fed two-hourly. When discharged from hospital at the age of one month, the baby was almost up to birth weight. He was receiving "Bengerized" cow's milk in the proportion of 1:1, with four teaspoonfuls of emulsion; three and a half ounces were given three-hourly. Food of high Calorie content was needed until he was aged five months, owing to his inability to tolerate large feeds, and "Benger's Food" proved very satisfactory. Mixed feeding was commenced at the age of five months. At the time of the meeting he appeared to be a normal, healthy baby.

The comment was made on the group of patients that the only symptom the three babies had in common was the vomiting of bile-stained fluid. In two cases that did not occur until the fifth day. In one case the stools were normal, and in another they were not sufficiently abnormal to cause concern. Any child suffering from vomiting in the first week of life should be carefully observed, and if bile was present in the vomitus the condition should be regarded as due to duodenal obstruction until proved otherwise. It was probable that prompt operation while the baby's condition was still good had been an important point in preserving the life of the child in the third case.

Exsanguination Transfusion for Hæmolytic Disease of the Newborn.

Dr. Isbister and Dr. Gaston presented a male child who had been treated by exsanguination transfusion for hæmolytic disease of the newborn. The mother had had four previous pregnancies, the first two ending in normal full-term births, the third and fourth ending in miscarriages, when blood transfusions were given. The transfusions were not given in a major hospital, and there seemed little doubt that at least one must have been given with Rh-positive blood. The mother's blood was typed before the birth of the child under discussion and was reported to be Rh-positive, so no tests for agglutinins were carried out and the mother attended a suburban antenatal clinic for the duration of a normal pregnancy. She was admitted to hospital in labour with hydramnios and was delivered without difficulty of a full-term infant weighing eight pounds three ounces in June, 1950. At birth the baby was jaundiced but pale with large petechial hæmorrhages on the face and limbs. The liver and spleen were enlarged almost to the region of the umbilicus, and the child's condition appeared serious. There was no œdema. The vernix was golden, and the *liquor amnii* was excessive in amount. The placenta, which weighed one pound ten ounces, was jaundiced. As the condition had not been anticipated, no cord blood had been collected for routine tests. The mother's blood was retyped and found to be Rh-negative. The baby's blood was Rh-positive and of group O. His hæmoglobin value was 13·5 grammes *per centum* (normal value 18 to 20 grammes *per centum*). The result of a Coombes test was positive. The total red blood cell count was 3,540,000 per cubic millimetre. The proportion of nucleated red cells was 78%, of myelocytes 3%, of metamyelocytes 5%, of band forms 6%, and of neutrophile cells 6%. An exchange transfusion by the umbilical route with Rh-negative female blood was started four hours after the child's birth. He was then much paler, the hæmorrhages had extended, and he appeared moribund. Five hundred millilitres were put in and 430 millilitres removed. Twitching of the face started during the transfusion. The next day the child's condition was poor, twitching of the face persisted for twenty-four hours, and continuous oxygen and sedative administration was needed. There was no head retraction or generalized convulsions. The hæmoglobin value was 13·3 grammes *per centum*. Food was being taken satisfactorily. On the fourth day there was an exacerbation of petechial hæmorrhages apparently controlled by the administration of five milligrammes of vitamin *K*. Jaundice was severe. The hæmoglobin value did not fall and was 16·2 grammes *per centum* on the twelfth day. The urine was very dark, and the stools were clay-coloured with no appearance of pigment before the child's discharge from hospital. During the third week a pustular rash developed which responded to penicillin therapy. On his discharge from hospital the child was aged one month and weighed seven pounds twelve and a half ounces. Jaundice was still present, the liver and spleen were slightly smaller than at birth, and the stools were clay-coloured. He was being fed on "Bengerized" cow's milk with methionine 0·175 gramme daily. The prognosis was considered to be bad, but the care and attention of a devoted mother seemed to offer

more than continued stay in hospital. For the next two months the child attended the pædiatric clinic, but progress was slow. He had severe attacks of colic and a voracious appetite. The colic was finally relieved by tincture of belladonna and was assumed to be biliary in origin. The appetite was met by an increase in the "Bengerized" cow's milk in strength and volume till the child was receiving twice his caloric requirements. The jaundice faded slowly. The liver and spleen were still easily palpable at the end of six months. At the end of three months bile pigments appeared in the stools, and the child began to gain weight rapidly, gaining nine pounds in four months. At the end of twelve months he had more than trebled his birth weight. At the time of the meeting he appeared to be making normal progress.

The comment was made that there appeared to be little doubt that the child would have died if treated by simple transfusion, as that could have corrected only the anæmia, which was not the only serious feature. Extensive liver damage and some signs of nervous involvement were evident. It was suggested that both liver necrosis and obstructive jaundice from bile pigments or debris had occurred and that regeneration of the liver was not adequate until the age of about six months. Up till that time a diet of high Calorie content was required mainly in the form of protein and carbohydrate.

A MEETING of the Victorian Branch of the British Medical Association was held at Greenvale Sanatorium, Greenvale, on July 16, 1951. The meeting took the form of a series of clinical demonstrations by members of the medical staff of the hospital.

Medical Management of Tuberculosis.

DR. MARGARET PLAYLE discussed a series of patients suffering from tuberculosis who had had medical treatment.

Three patients with advanced tuberculous infection had improved greatly with routine sanatorium rest and exercise. All were now very well and active.

Five patients, admitted to hospital with very advanced infection throughout both lungs, had been given PAS and streptomycin. All had still a good deal of infiltration, but it was much lessened, and the general condition of each was excellent.

Five patients, who had had minimal infection on admission to hospital, had deteriorated despite all treatment. Four had died, and the fifth was undergoing thoracoplasty.

Four patients with cavitation had been relieved by means of phrenic crush and pneumoperitoneum, and all were now very well and active.

Two young patients, aged thirteen and sixteen years respectively, had made remarkable recovery; each had had a large degree of cavitation. One had obtained complete recovery with streptomycin therapy and the other with streptomycin therapy, artificial pneumothorax and, finally, phrenic nerve crush.

Methods of Diagnosis of Tuberculosis.

A demonstration was prepared showing some of the methods for the diagnosis of tuberculosis in routine use at the public health bacteriological laboratory in the University of Melbourne.

The exhibits included a comparison of Ziehl-Neelsen and fluorescence microscopy and the seven-day method for assessing drug sensitivity of tubercle bacilli; an explanation was given of their underlying principles by Dr. M. M. Wilson and Mr. L. J. Swaby.

Physiotherapy and Thoracic Surgery.

MISS A. E. T. HOOPER and MISS B. GERAND, members of the Australian Physiotherapy Association, showed several patients who had undergone thoracoplasty and pneumonectomy. The following aspects of pre-operative training of the patients were explained: (i) the teaching of diphragmatic breathing with relaxed and controlled upper section of the chest; with resection—either pneumonectomy or lobectomy—localized expansion of the bases was added; (ii) corrective exercises for possible post-operative deformities—especially the lateral deviation of the head high shoulder and the long lean of the body; (iii) arm exercises for shoulder joint and shoulder girdle.

The application of these methods was demonstrated with the patients presented. They showed good control of their breathing, good posture and minimum scoliosis, and a full range of arm movement.

It was then explained that treatment was commenced the day after operation with (i) assistance in maintaining good posture, particularly of the head, (ii) diaphragmatic breathing to aid coughing, overcome breathlessness and prevent paradoxical movement of the upper part of the chest, (iii) localized basal expansion in cases of resection, and (iv) a full range of shoulder movements. Treatment progressed each day with gradually increasingly active movements for head and arm. Normally there was a full active range of arm movements ten days after each stage. Treatment was continued daily for about four weeks after operation, and then at less frequent intervals. Mirrors were always used as an aid in the correction of posture.

X-ray films were shown contrasting the gross scoliosis of untreated patients who had undergone thoracoplasty and the straight spines of the patients who had received the treatment as demonstrated.

It was explained that the treatment demonstrated was based on the principles in use at the Brompton Chest Hospital in London, where Miss Hooper has recently been attached to the staff.

General.

A display was available for inspection of patients' rules, staff ward rules, menus and the patients' library catalogue.

MISS H. SHANAHAN, chief nurse of the Central Tuberculosis Bureau, discussed domiciliary management of patients by her staff of visiting nurses.

At the close of the meeting, to illustrate activities within the sanatorium, visitors were entertained by a short two-act play, presented by patients, and by a "Technicolor" movie film, which had been prepared specially for new patients and for patients receiving bed-rest over a prolonged period, to convey to them activities and recreational facilities beyond the wards. The film showed glimpses of the library and conveyance of books to bed patients in the wards, typewriting, letter writing, entertainment by jig-saw and crossword puzzles, and the handcraft room with girls doing needlework, rug making, plastic plaiting and all branches of dressmaking, cutting out, fitting, machining and pressing. The film also showed preparation of food in the kitchen.

Medical Societies.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held in the Anatomy Lecture Theatre, Frome Road, Adelaide, on September 7, 1951.

Liver Aspiration Biopsy in the Study of Iron Metabolism.

MR. H. J. LEE presented a communication on the use of liver aspiration biopsy in the study of iron metabolism in sheep. He described A. T. Dick's method for withdrawing liver samples from living sheep by passing a stainless steel cannula of four to five millimetres' internal diameter right through the liver and demonstrated the apparatus. He outlined the hazards involved and went on to say that by this procedure representative samples of liver which usually weighed between 100 and 200 milligrammes (D.M.) were obtained from individual sheep on many occasions, at short intervals if necessary. Such samples proved ample for the estimation of copper and iron concentrations in liver. Experimental evidence indicated that the chances of contamination of the liver samples by iron from the instruments were negligible, and also that variable distribution of iron throughout the liver or errors introduced by the inclusion of variable amounts of blood within the samples would not be sufficient to invalidate the conclusions drawn from analytical results.

A study was made of the iron and copper concentrations in the livers of sheep whose diet was (i) acutely copper deficient, (ii) inadequately supplemented with copper and (iii) adequately supplemented with copper; and in the livers of sheep whose copper status had been restored after a prolonged period of acute copper deficiency. A reciprocal relationship was shown to exist between iron and copper to the extent that when the copper concentration fell below 10 parts per million (D.M.), the iron concentration rose from the usual level of 100 to 300 parts per million to levels as great as 20,000 parts per million. At least part of this iron

was stored as hæmosiderin. Acutely copper-deficient sheep developed an anæmia which could be quickly overcome by dosing with copper. The excessive accumulations of iron in the livers of these animals were gradually dissipated when the copper status was restored and eventually achieved normal levels after about two years. The stored iron was evidently not used for the synthesis of hæmoglobin, as the iron concentrations did not fall appreciably until some months after the anæmia had been corrected.

Mr. Lee summarized current views on iron metabolism and stressed the widely held contention that iron, once absorbed by the body, was never excreted. Despite this view, it was tentatively suggested that the excess iron probably had been excreted by the experimental sheep under discussion. It was considered that redistribution of iron stored in the liver amongst other tissues was unlikely, although that possibility had still to be checked.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

CERTIFICATE OF EXAMINATION OF WILLIAM REDFERN.[1]

[*Historical Records of Australia*, September 6, 1808.]

We whose Names are hereunto subscribed do hereby certify that We have examined Mr. William REDFERN touching his Skill in Medicine and Surgery and the other necessary Collateral Branches[2] of Medical Literature, and that We find him qualified to exercise the Profession of a Surgeon &c. And consequently to fill the Situation of an Assistant Surgeon in any Department of His Majesty's Service.

Given under Our Hands at Sydney in New South Wales this first day of September 1808.

Thos. Jamison, Principal Surgeon.
J. Harris, Surgeon, New So Wales Corps.
Wm. Bohan, Assistant Surgeon, New South Wales Corps.

Correspondence.

A CASE OF DEATH FOLLOWING SECTION, LIGATION AND INJECTION OF VARICOSE VEINS.

Sir: Dr. A. E. Coates is to be congratulated for reporting this patient (The Medical Journal of Australia, September 22, 1951). Such a happening after an operation for varicose veins is, indeed, tragic, and the report of such an event should stimulate thought as to the possible cause so that future tragedies might be prevented.

Certain features of Dr. Coates's report call for comment. The operation of saphenous vein section and retrograde injection which was done on this patient is referred to as the "simplest routine surgical procedure". The difficulties of technique to obtain a cure are obvious from the bad results which are so common; the risks are obvious from the reported—and unreported—complications. As long as this operation is regarded as a minor operation or simple routine procedure, so often will bad results and mishaps occur.

At the operation performed by Dr. Coates, two millilitres of "Ethamolin" were injected in the lower third of the leg at the lowest point of section of the saphenous vein. Full strength "Ethamolin" is a dangerous substance to use in conjunction with ligation and section of a vein. It should never be used—particularly in a limb that has been previously the site of a deep vein thrombosis.

Phlebographic studies show that material injected into the lower end of the long saphenous vein very readily enters the deep system, as there is an anastomosis between the two systems near the ankle joint. An injection, moreover, given in this region would have a very short distance to travel before reaching the deep veins, and so very little dilution with blood would occur. Thrombosis in the calf veins could then take place. This actually did happen, as the post-mortem examination showed. As the patient was ambulatory from the time of the operation, and as the fatal embolus occurred so early, suspicion must fall on the injection. Dr. Coates states that he has long since given up injection of sclerosing substance into the upper part of the veins, but he does not state why. If his reason is based on phlebographic studies of Boyd and Robertson, I would point out that their conclusions are unsound.

This subject has been fully discussed recently (Lawes, 1951).

I do not write this letter as a criticism of Dr. Coates, to whom go our sympathies in this matter. I feel, however, that one should go further than humility in the face of "slings and arrows of outrageous fortune". It may have been so, but my point is that such happenings should be closely studied in the hope that ways may be found to prevent recurrences.

Yours, etc.,
Undated. C. H. Wickham Lawes.

Reference.

Lawes, C. H. Wickham (1951), "Some Points in the Management of Varicose Veins", The Medical Journal of Australia, Volume I, page 637.

A DISCLAIMER.

Sir: We wish to assert categorically that the use of our names in articles appearing in the lay Press in Sydney on September 25 and October 10 was entirely without our knowledge or consent and without the knowledge or consent of the Public Health Department of Western Australia. We wish to dissociate ourselves entirely from any such unauthorized publicity.

Yours, etc.,
W. E. Gye.
Ida Mann.
Perth,
October 12, 1951.

SOME OBSERVATIONS ON THE TREATMENT OF OTITIS EXTERNA AND OTITIS MEDIA.

Sir: I was interested to read the comments on seborrhœic dermatitis of the scalp in the very interesting article "Some Observations on the Treatment of Otitis Externa and Otitis Media", by W. E. David, M.C., M.B., Ch.M., in The Medical Journal of Australia, September 29, 1951.

The reference to seborrhœa of the scalp as a causative factor of *otitis externa* is particularly interesting, and I have recognized it as such for some years now. I have thought that the medical profession should give this condition more attention and that the public health authorities should insist on stricter sterilizaion of the barbers' implements. In spite of Dr. David's statement that the clippers cannot be sterilized, I think something may be done in this direction. Perhaps an antiseptic spirit for the instruments or ultra-violet light could be successful. At least something should be attempted, and there may be more efficient methods, because the condition is very prevalent and mainly due to this source of infection.

Incidentally, the mention of ultra-violet rays suggests a very effective treatment for *otitis externa* not mentioned by Dr. David. This is applied with a Kromayer lamp and quartz applicator, and one or two applications are usually sufficient to cure this complaint in most of its types. If seborrhœa of the scalp is the cause, this must be cleared up as well with suitable treatment.

I have noticed that many patients with chronic nasal infection have quite a degree of seborrhœa of the scalp. This may be quite coincidental, but I should think that if the infection can enter the external auditory canal and infect it, it could as likely do the same to the nasal passages and sinuses. One can visualize the head on the pillow at night and the intake of air on inspiration through the

[1] From the original in the Mitchell Library, Sydney.

[2] The examination of William Redfern and that of Edward Luttrell, which was held at the same time, were probably the first medical tests held in the Colony. This system was subsequently extended to an examination of all who commenced practice in the Colony. Anyone failing to pass the examination was gazetted and ordered to desist from practice.

nostrils facilitating this process of infection. Thus treatment of the scalp in these cases could be a distinct help in their treatment.

Yours, etc.,
T. W. BURGESS.

135 Macquarie Street,
Sydney,
October 3, 1951.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 73, of September 27, 1951.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Resignation.—The resignation of Bertram Charles Morgan of his appointment as Surgeon Lieutenant (for short service) is accepted, dated 17th July, 1951.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Grant of Honorary Rank.—John O'Sullivan is granted the honorary rank of Surgeon Commander, dated 12th July, 1951.

ROYAL AUSTRALIAN AIR FORCE.

Air Force Reserve: Medical Branch.

The following are appointed to commissions with the rank of Flight Lieutenant: Robert William Edward Manser (257479), 2nd July, 1951, Vennard Francis O'Neill (018091), 14th May, 1951, William Eugene Downey (257819), 23rd May, 1951, Donald Millis McLean (257820), 12th June, 1951.

The appointments of the following Flight Lieutenants are terminated: R. K. Smyth (257715), 30th November, 1945, B. E. Brookman (287468), 16th May, 1951.

Notice.

AUSTRALASIAN ASSOCIATION OF PSYCHIATRISTS.

THE following is the programme of clinical sessions at the meeting of the Australasian Association of Psychiatrists in Sydney. All members of the British Medical Association are invited to attend.

October 30, 1951: 8.15 p.m., in the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, "Termination of Pregnancy on Psychiatric Grounds", Dr. C. M. Macarthy. Discussion will be opened by Dr. C. Swanton and Dr. J. A. McGeorge.

October 31, 1951: 11 a.m., clinical demonstration at the Repatriation General Hospital, Concord; 2.30 p.m., neurological demonstration at the Royal Prince Alfred Hospital, Camperdown.

November 1, 1951: 2.30 p.m., in the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, "Transference and Counter Transference in Psychoanalytical Therapy", Andrew Peto (M.D., Budapest). Discussion will be opened by Dr. F. W. Graham and Dr. A. R. Phillips.

Medical Appointments.

Dr. E. F. Gartrell has been appointed an honorary physician in the Royal Adelaide Hospital.

Dr. P. P. Bateman has been appointed an honorary clinical assistant to the allergy clinic in the Royal Adelaide Hospital.

Dr. R. G. Plummer has been appointed an honorary assistant aural surgeon in the Royal Adelaide Hospital.

Dr. Alfreda Wilma Thrush has been appointed refractionist to the Royal Adelaide Hospital.

Dr. C. A. T. Edwards has been appointed public vaccinator for the Shire of Numurkah, Victoria.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED SEPTEMBER 22, 1951.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	..	..	..
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	1	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	..	1	..	..	..	..	..	1
Diphtheria	3(1)	2(2)	3	1	4(4)	..	..	..	13
Dysentery (Bacillary)	..	1(1)	..	..	2(2)	..	..	..	3
Encephalitis	..	1	..	..	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	..	..	..	..	9(7)	..	..	..	9
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	1	..	1
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	1(1)	..	..	..	..	..	1
Meningococcal Infection	4(3)	3(1)	1	1(1)	2	..	1	1	13
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	15(4)	5(2)	4	29(21)	2(1)	..	..	..	55
Puerperal Fever	..	..	..	1(1)	..	..	..	..	1
Rubella	..	20(11)	1	..	3(2)	..	..	..	24
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	19(12)	10(7)	4(3)	7(7)	1(1)	..	..	..	41
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	1	..	..	..	2
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	28(18)	10(7)	24(17)	9(8)	7(6)	4	2	..	84
Typhoid Fever	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne)	..	..	..	..	..	..	..	..	..
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Dr. R. de Garis Bernard has been appointed senior honorary assistant radiotherapist in the Royal Adelaide Hospital.

Dr. J. MacL. Gooch has been appointed public vaccinator for the city of Sale, Victoria.

Dr. H. A. Retallick has been appointed public vaccinator for the Shire of East Loddon, Victoria.

Dr. G. J. Kennedy has been appointed public vaccinator for the Shire of Tungamah, Victoria.

Dr. M. Hoban has been appointed public vaccinator for the Shire of Beechworth, Victoria.

Dr. W. J. Moon has been appointed public vaccinator for the Shire of Kerang, Victoria.

Dr. O. P. Burger has been appointed public vaccinator for the Shire of Corio, and for the city of Geelong, Victoria.

Dr. G. H. Way has been appointed public vaccinator for the Shire of Deakin, Victoria.

Dr. D. J. Bartram has been appointed public vaccinator for the Shire of Waranga, Victoria.

Dr. J. M. Thompson has been appointed honorary pathologist and bacteriologist to the Port Pirie Hospital, South Australia.

Dr. C. R. D. Brothers has been appointed a member and deputy chairman of the Mental Hygiene Authority of Victoria.

Dr. W. F. H. Crick has been appointed a public vaccinator for the Borough of Stawell, Victoria.

Dr. A. A. Gray has been appointed a public vaccinator for the Shire of Otway, Victoria.

Dr. W. J. Alexander has been appointed a public vaccinator for the Shire of Benalla, Victoria.

Dr. R. C. Angove has been appointed honorary clinical assistant to the electrocardiograph department in the Royal Adelaide Hospital.

Dr. R. A. Burston has been appointed assistant medical superintendent in the Royal Adelaide Hospital.

Dr. J. A. Earl has been appointed medical registrar in the Royal Adelaide Hospital.

Dr. G. B. Fisk has been appointed gynæcological registrar in the Royal Adelaide Hospital.

Australian Medical Board Proceedings.

QUEENSLAND.

THE undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, as duly qualified medical practitioners:

Ganz, Lazar, M.B., B.Ch., B.A.O., 1942 (Queen's Univ., Belfast), M.R.C.P. (Ireland), 1943, F.R.C.S. (Edinburgh), 1950, Atcherley Hotel, 513 Queen Street, Brisbane.

Deane, Joseph Eugene, L.Med.L.Ch., 1923, M.B., B.Ch., 1925 (Trinity College, Dublin), Margate Parade, Margate.

Stranger, Donald James, M.B., B.S., 1944 (Univ. Sydney), Texas.

Phillips, John Bertram, M.B., B.S., 1946 (Univ. Sydney), Thursday Island Hospital, Thursday Island.

The undermentioned has been registered, pursuant to the provisions of *The Medical Acts*, 1939-1948, as a specialist in medicine:

Aaron, Kurt, 905 Stanley Street, East Brisbane, M.R.C.P. (Edinburgh), 1951.

Obituary.

JOHN SANDISON YULE.

WE regret to announce the death of Dr. John Sanderson Yule, which occurred on September 27, 1951, at East Brighton, Victoria.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Fenwick, Louis, M.B., B.S., 1950 (Univ. Sydney), Sydney Hospital, Macquarie Street, Sydney.

Ferguson, Edward Stephen Perry, M.B., B.S., 1950 (Univ. Sydney), 20 Beach Road, Collaroy.

Leitch, David Stanley, M.B., B.S., 1950 (Univ. Sydney), "Springville", Narrabri.

Dickson, John Grant, M.B., B.S., 1944 (Univ. Sydney), c.o. Mr. E. W. Statham, Adam's Chambers, 195 Elizabeth Street, Sydney.

Egan, James Edward, M.B., B.S., 1947 (Univ. Sydney), Wellington Road, Chester Hill.

Diary for the Month.

Oct. 23.—New South Wales Branch, B.M.A.: Ethics Committee.
Oct. 24.—Victorian Branch, B.M.A.: Council Meeting.
Oct. 25.—New South Wales Branch, B.M.A.: Branch Meeting.
Oct. 25.—South Australian Branch, B.M.A.: Branch Meeting.
Oct. 26.—Queensland Branch, B.M.A.: Council Meeting.
Nov. 1.—South Australian Branch, B.M.A.: Council Meeting.
Nov. 2.—Queensland Branch, B.M.A.: Branch Meeting.
Nov. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 7.—Western Australian Branch, B.M.A.: Council Meeting.
Nov. 9.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £4 *per annum* within Australia and the British Commonwealth of Nations and £5 *per annum* within America and foreign countries, payable in advance.